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The effects of pre-cooling on skin blood flow during exercise in the heat and subsequent performance

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**The Effects of Pre-Cooling on Skin Blood Flow
During Exercise in the Heat and Subsequent
Performance**

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MScR Exercise Physiology

2012

The Effects of Pre-Cooling on Skin Blood Flow During Exercise in the Heat and Subsequent Performance

By Matthew Maley

A thesis submitted in partial fulfilment of the University's requirements for the
Degree of Master of Research.

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Abstract

Introduction: Exercise disturbs the homeostatic state of the human body causing an increase in heat production which is exacerbated in hyperthermic conditions. Ice vest cooling both before and during exercise has been shown to alleviate thermal strain and attenuate a rise in core temperature. It has been proposed that skin blood flow may be significantly reduced during cooling and blunted during subsequent exercise with increases in skin blood flow, due to exercise, cooling the perfusing blood which abate a rise in core temperature on return.

Aims: The purpose of this study was to determine whether pre-cooling (COOL), with an ice vest, reduces skin blood flow compared to a no cooling control (CON), and whether subsequent exercise and 3-km performance times in the heat are improved on a cycle ergometer. A secondary aim was to determine the effects of ice vest cooling on the physiological responses during seated rest and heat exposure to observe the responses of cooling without exercise metabolic heat production.

Methods: Eight male participants volunteered to take part in the study. The mean (\pm SD) age, height, weight and maximal oxygen uptake ($\dot{V}O_{2\max}$) were; 24.5 ± 5.0 years, 178.7 ± 2.6 cm, 77.5 ± 13.7 kg, and 43.4 ± 8.6 ml.min.⁻¹kg.⁻¹, respectively. Ice vest cooling was applied in cool conditions for 20 min prior to exercise and seated rest in the heat ($35.4 \pm 0.4^{\circ}\text{C}$, $26.3 \pm 4.1\%$ RH). The intermittent exercise protocol was carried out on a cycle ergometer and consisted of nine \times five min bouts of exercise consisting of; 2 min 30 seconds at 40% $\dot{V}O_{2\max}$, 1 min 30 seconds at 60% $\dot{V}O_{2\max}$, 30 seconds at 100% $\dot{V}O_{2\max}$, and 30 seconds of unloaded cycling at a cadence of 70 rev.min.⁻¹. Following this, a three kilometre performance trial (PT) was performed at a resistance set at 60% $\dot{V}O_{2\max}$ with the instruction to complete the distance as fast as possible. Seated rest in the heat was undertake for 45 min. Rectal (T_{rec}) and aural (T_{aur}) temperature, mean skin temperature (T_{ms}), skin blood flow (SkBF),

heart rate (HR), oxygen uptake (VO_2), blood lactate ([BLa]), perceived exertion (RPE), thermal strain (RPTS), and profile of mood states (POMS) were recorded throughout the trial. A two-way analysis of analysis of variance (ANOVA) with repeated measures on both factors (trial \times time) was used to determine statistical differences.

Results: SkBF was not significantly affected by ice vest cooling at any time point during cooling, seated rest in the heat or exercise trials ($P>0.05$). No significant interactions were observed during cooling for both T_{aur} and T_{rec} ($P>0.05$). T_{aur} throughout the seated rest trial ($P<0.01$) remained significantly lower following a cooling period compared to CON. T_{aur} was significantly lower from 5-25 min of exercise ($P<0.01$) following a cooling period compared to CON. T_{rec} was significantly lower at the end of the seated rest trial in the heat following a cooling period compared to CON ($P<0.01$) but did not differ at any time point during the exercise trial ($P>0.05$). T_{ms} and RPTS was significantly lower during cooling compared to CON ($P<0.01$) but did not differ thereafter. No significant difference was observed for performance between trials.

Discussion: It appears that the attenuated rise in core temperature is not due to a blunted rise in SkBF during exercise. It is possible that following cooling there is a significant heat sink created at the skin which cools the perfusing blood in the cutaneous circulation which would cool the core on return. Despite this, there were no physiological or POMS data to demonstrate that ice vest cooling improved exercise in hyperthermic conditions. As it was shown in the present study T_{rec} only significantly differed after 45 min of seated rest in the heat following cooling, therefore future research should assess the optimum post cooling time for which exercise should begin.

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1.0 Introduction

Humans have an ability to maintain a relatively stable core temperature of $37 \pm 1^\circ\text{C}$ despite varying ambient temperatures (Gleeson 1998). This thermal homeostasis is attributable to the ability of the body to match heat production with heat loss (Blatteis 2001). However, exercise and exposure to hyperthermic conditions further challenges the thermoregulatory system. As a result core temperature will increase due to the reduction in heat dissipation particularly when exposed to hot humid environments (Wendt, van Loon, and van Marken Lichtenbelt 2007). Where the evaporative capacity of the environment is unable to remove heat being produced by the body this results in an uncompensable heat stress (Robinson and Gerking 1947, Latzka et al. 1998) which can lead to a reduced exercise performance and an increased risk of heat illness (Hargreaves 2008).

During exercise skin blood flow and sweat rate increase (Casa et al. 2010, Rowell 1974), which result in a loss of heat from the blood to the skin which is subsequently lost to the environment due to a thermal gradient. The cooler cutaneous blood, from the skin, then returns to the core in an attempt to abate a rise in core temperature and maintain a thermal balance. In addition to the physiological mechanisms of heat loss there are various strategies that an exercising individual can utilise in an attempt to attenuate the increase in core and body temperature and reductions in performance associated with exercise in hyperthermic conditions. These include pre-cooling (Bogerd et al. 2010), pre and mid-event cooling (Price, Boyd, Goosey-Tolfrey 2009), and acclimation/acclimatisation (Lorenzo et al. 2010). Specifically, pre-cooling can be used to offset the increase in body temperature during exercise in the heat by reducing core and skin temperature prior to exercise. The pre-cooling thereby increases the margin for metabolic heat production (Marino 2002). It has been

suggested that pre-cooling may initially cause cutaneous vasoconstriction due to the immediate cold stimulus (Wilson et al. 2007, Price, Boyd, and Goosey-Tolfrey 2009). Thereafter, as body temperature increases during exercise in the heat, the demand for skin blood flow would increase and as a result the blood perfusing the pre-cooled skin would be subsequently cooled and abate a rise in core temperature on return. Bogerd et al. (2010) measured skin blood flow pre and post cooling with a laser-Doppler technique and observed reductions in areas such as the back, chest, shoulder and fingers during pre-cooling as well as rectal temperature being reduced by 0.1°C. Consequently, there was an increased time to exhaustion observed following cooling compared to a control during cycling at 65% $\text{VO}_{2\text{peak}}$. Bogerd et al. (2010), however, did not measure skin blood flow during exercise in the heat which would have provided more insight into an area that lacks a definitive mechanism by how pre-cooling facilitates exercise performance in the heat. Therefore, the aim of this study was to assess skin blood flow in addition to other physiological and psychological responses during pre-cooling and exercise performance in hyperthermic conditions. This will hopefully further our understanding of how pre-cooling may facilitate exercise performance under such conditions. An additional aim of this study was to assess the physiological and psychological responses of a cooling period prior to seated rest in the heat as this would be useful in identifying the responses to cooling without an exercise stimulus.

2.0 Literature Review

Core temperature in humans is maintained within a narrow range ($37 \pm 1^{\circ}\text{C}$) which is necessary for efficient bodily functions (Blatteis 2001). Thermal balance requires heat gain from endogenous (metabolic) and exogenous (environmental) sources to be dissipated through conduction, convection, radiation and evaporation (Cheuvront and Haymes 2001). Exercise causes this thermal balance to tip in favour of heat gain and is thought that hyperthermia is one of the dominant factors contributing to the onset of fatigue during exercise in high environmental temperatures (Tatterson et al. 2000). The following literature review will discuss how the body regulates temperature through alterations in cutaneous blood flow and sweat rate during exercise in normothermic and hyperthermic conditions. In addition, studies assessing physiological limitations to exercise in the heat and how pre-cooling is utilised in an attempt to limit the debilitating effects of hyperthermic conditions on exercise performance will be reviewed.

2.1 Temperature Regulation

The rise in core temperature during exercise is a result of the inefficiency of skeletal muscle metabolism as more than 75% of the energy that is generated by skeletal muscle substrate oxidation is liberated as heat (Sawka 1992). The rise in muscle temperature leads to a reversal of the temperature gradient between muscle and arterial blood. Heat is therefore transferred down this thermal gradient from muscle tissue to blood and then to the core (Wendt, van Loon, and van Marken Lichtenbelt 2007). Hypothalamic centres of the brain will detect such incoming afferent information which would lead to an appropriate efferent response.

2.1.1 Role of the Hypothalamus

Lesion and stimulation studies suggest that no one single neural area acts as the centre of thermoregulation instead there appears to be a hierarchy of structures extending throughout the hypothalamus, brain stem and spinal cord (Boulant 2000). Lower brain stem and spinal structures are capable of detecting changes in body temperature and initiating thermoregulatory responses but higher structures, such as the preoptic nucleus and anterior hypothalamus, regulate body temperature more precisely (Mekjavic and Eiken 2006). The preoptic-anterior hypothalamus monitors the temperature of the blood perfusing the brain and thus can detect changes in core temperature. In addition to this, the hypothalamus also receives afferent information from thermoreceptors throughout the body including abdominal viscera, spinal cord and the skin (Rawson, Quick, and Coughlin 1969, Charkoudian 2003, Wendt, van Loon, and van Marken Lichtenbelt 2007). Comparisons can then be made from afferent information between the central and peripheral temperatures which lead to an appropriate efferent response in an attempt to bring core temperature back to its 'set point' (Cheung, McLellan, and Tenaglia 2000). Subsequently, it is a proportional control system with efferent responses dependant on the magnitude of the afferent information (Mekjavic and Eiken 2006).

2.1.2 Core Temperature Measurements

Indirect measures of core temperature are generally used to estimate the temperature for the hypothalamus. There are various methods that can be utilised to measure core temperature which include; rectal, aural, tympanic, oesophageal and pulmonary artery temperatures. Pulmonary artery temperature has been suggested to provide the most accurate reading of core body temperature (Fulbrook 1997). However, it is deemed unsafe and not practical during exercise. Aural (T_{aur}) and tympanic (T_{tyl}) temperature have the advantage of being

easily accessible during exercise. In addition, the tympanic membrane is supplied by blood from the carotid artery which also supplies blood to the hypothalamus which makes measurement at the ear a practical and accurate method (Bricknell 1997). There is though concern over the effect of the external environment on T_{aur} but this can be overcome with cotton wool insulation. Oesophageal temperature (T_{oes}) when correctly positioned is in close proximity to the aorta which is why this method has been frequently utilised and validated (Nybo and Nielsen 2001). However, this method can provide discomfort to an exercising individual. Finally, rectal temperature (T_{rec}) appears to be utilised most frequently in research studying thermoregulation. This is due to the accuracy of T_{rec} as it has been found to correlate well with pulmonary artery temperature (Fulbrook 1997). However, T_{rec} is notoriously known for the “lag” in response to change in temperature due to its relatively large mass compared to that of the ear canal for T_{aur} or T_{tym} (Gagnon et al. 2010). Despite this, Gass and Gass (1998) compared T_{rec} and T_{oes} temperature before and after 36 min of continuous exercise ($65\% \dot{V}O_{2max}$) on a treadmill, arm and cycle ergometer. By the end of exercise only a 0.2°C difference between locations was observed, suggesting T_{rec} is an accurate and valid measurement of core temperature.

2.1.3 Heat Exchange

For heat loss to occur excess heat needs to be transferred from the core to the skin where heat can be dissipated to the environment (Wendt, van Loon, and van Marken Lichtenbelt 2007). The rate of heat transfer from core to skin is determined by the thermal gradient between the two, with a larger temperature gradient leading to greater heat loss. In addition, an increase in the cutaneous vasodilator system is required to increase skin blood flow to further enhance this heat transfer from the core to the skin where it can be dissipated to the environment. When environmental temperature reaches approximately 35°C there is an increased

dependency on evaporative heat loss as the thermal gradient between the skin and environment is reduced (Kruk et al. 1991, Nelson 1938 cited in Powers and Howley 2007: 250). Activation of eccrine sweat glands causes sweat to be secreted onto the skin surface (Shibasaki, Wilson, and Crandall 2006) with evaporation of one litre of sweat removing 2.4MJ of heat from the body (Gleeson 1998). Once heat has been transferred to the skin there are four ways by which heat can be dissipated to the environment; radiation, conduction, convection and evaporation (Cheuvront and Haymes 2001). The heat exchange pathway consequently has an effect on the net heat storage of the body. When net heat storage is positive body temperature will subsequently rise and vice versa. The following sections present key effector mechanisms enabling heat exchange to occur.

2.1.4 Control of Cutaneous Circulation

Cutaneous circulation performs a primary role in the regulation of body temperature through control of the level of perfusion. Blood flow perfusing the skin is influenced by both thermoregulatory (core and skin temperature) and non-thermoregulatory reflexes (e.g. baroreflex) (Hodges and Johnson 2009). Skin blood flow at rest in normothermic environments is approximately $250 \text{ ml} \cdot \text{min}^{-1}$ (Johnson 1992, Rowell 1974) and is increased several fold during exercise which substantially increases convective transfer of heat from the core to the skin (Charkoudian 2003). This higher demand for cutaneous perfusion and active skeletal muscle perfusion during exercise often requires an increased heart rate (HR), stroke volume and vasoconstriction in the splanchnic regions (Kenney and Johnson 1992). However, a vasoconstrictor response in skin blood flow is noticeable at the initial onset of exercise due to an exercise-induced vasoconstrictor effect (Kellogg, Johnson, and Kosiba 1991) but is quickly withdrawn and overridden by active cutaneous vasodilation to facilitate heat dissipation (Kenney and Johnson 1992).

Skin blood flow is controlled by two arms of the sympathetic nervous system; an adrenergic vasoconstrictor system and a vasodilator system (Kenney and Johnson 1992). Figure 2.1 shows an overview of these systems. Changes in core and skin temperature provide afferent information to the hypothalamus which produces an appropriate efferent response that includes alteration of sympathetic vasoconstrictor nerves and active vasodilator nerves. Local cooling initiates localized neurotransmission from noradrenergic nerves which release norepinephrine (Taddei, Pedrinelli, and Salvetti 1990) and neuropeptide Y (Nilsson et al. 1996) to cause vasoconstriction (Charkoudian 2003).

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Figure 2.1. Overview of thermoregulatory control of cutaneous circulation (Minus signs refer to inverse relationships whereas plus signs refer to positive relationships vice versa) (Charkoudian 2003: 606). NE= Norepinephrine, NPY= Neuropeptide Y, CGRP= calcitonin gene-related peptide, SP= substance P, NKA= neurokinin A, NO= nitric oxide.

Active sympathetic vasodilator nerves release an unknown neurotransmitter to cause vasodilation (Charkoudian 2003, Kenney and Johnson 1992). In addition, increases in local

temperature cause vasodilation by stimulating local neuropeptide release from sensory nerves mainly from calcitonin gene-related peptide (Jager et al. 1990), with smaller contributions from substance P (Weidner et al. 2000) and neurokinin A (Pedersen-Bjergaard et al. 1991). Nitric oxide also causes vasodilation and is important in both the initiation and the maintenance phase of vasodilation (Minson, Berry, and Joyner 2001).

Greater increases in skin blood flow are required in order to facilitate the dissipation of the greater heat storage during exercise in hyperthermic conditions (Cheuvront and Haymes 2001). Strenuous exercise in hyperthermic conditions can lead to increases in skin blood flow to $\sim 8 \text{ L}\cdot\text{min}^{-1}$ (Rowell 1974) which is $\sim 1\text{-}2 \text{ L}\cdot\text{min}^{-1}$ above that observed in normothermic conditions (Nielsen 1993, Ho et al. 1997). The large increase in skin blood flow is facilitated primarily, 80-90%, via the activation of sympathetic vasodilator nerves in the skin with the remaining 10-20% resulting from a withdrawal in the adrenergic vasoconstrictor system (Hodges and Johnson 2009). Skin blood flow rises linearly with increases in core temperature until an T_{oes} of approximately 38°C is reached which results in a plateau in skin blood flow (Bregelman et al. 1977). This plateau of skin blood flow has been found to be a consequence of the limitation of active vasodilation activity (Kellogg et al. 1993). A possible baroreflex response may be limiting vasodilation to cutaneous circulation in order to maintain mean arterial blood pressure in the face of active skeletal muscle vasodilation during exercise (Kellogg, Johnson, and Kosiba 1990).

2.1.5 Control of Sweating

Eccrine sweat glands are distributed over most of the body surface with between 1.6 and 4.0 million sweat glands in human skin (Kondo et al. 1998). Typical sweat rates during exercise can vary largely depending on exercise intensity and duration, environmental temperature and

hydration status of an exercising individual. Drust, Reilly, and Cable (2000) observed sweat rates of approximately $1.0 \text{ L} \cdot \text{min}^{-1}$ during an intermittent football specific protocol ($\sim 68\% \dot{V}O_{2\text{max}}$) under normothermic conditions (18°C). However, sweat rates as high as $2 \text{ L} \cdot \text{min}^{-1}$ have been observed during running at 27°C (Casa et al. 2010). It appears that sweating is proportionally controlled by brain temperature and secondly affected by mean skin temperature (Nadel et al. 1971). It has been observed that although mean skin temperature affects sweating via central mechanisms local temperature of the sweat gland can also influence sweat rate (Van Beaumont and Bullard 1965). Other influences such as baroreceptors, muscle mechanoreceptors, and metaboreceptors are known to fine tune the sweat rate of an individual which is also determined by hydration status (Shibasaki, Kondo, and Crandall 2003, Shibasaki, Wilson, and Crandall 2006).

2.2 Thermoregulation in Cool Conditions

Measurements of T_{rec} and T_{aur} temperature in normothermic environments ($19\text{--}25^\circ\text{C}$) have been shown to increase between $0.6\text{--}0.8^\circ\text{C}$ above resting values and plateau after approximately 30 min during exercise at $50\text{--}60\% \dot{V}O_{2\text{max}}$ (Nielsen et al. 1971, Powers, Howley, and Cox 1982). This plateau in core temperature is due to the body being in balance of heat gain and heat loss which demonstrates an 'adjustable set point' during exercise (Cabanac 2006). At greater exercise intensities, such as $70\% \dot{V}O_{2\text{max}}$, for a prolonged duration, core temperature has been shown to rise by up to 2°C above resting values (Parkin et al. 1999, Sawka, Knowlton, and Critz 1979) with no plateau observed. This is attributable to the inability of the body to match heat loss with heat gain, due to the higher intensity exercise causing greater metabolic heat production, resulting in core temperature continually rising to a critical point ($\sim 39^\circ\text{C}$). Core temperatures that are observed between $39\text{--}40^\circ\text{C}$ can

be critical to exercise performance as there is a reduction in the central drive to the working muscles which results in a reduction in exercise performance (Nybo and Nielsen 2001).

Skin temperature is generally maintained between 30–33°C (Gonzalez-Alonso et al. 1999, Rasch et al. 1991) but varies widely across the body and is often warmest in the central areas due to its proximity to the core (Clark 1981). Various studies have observed that exercise intensities between 60-75% $\dot{V}O_{2\max}$ in normothermic conditions produce an increase in mean skin temperature up to 3°C above resting values (Kenny et al. 1999, Wilson et al. 2002). This difference in temperature between the core and skin allows for heat to be transferred down the thermal gradient. To facilitate heat dissipation at the skin an increase in cutaneous vasodilation and sweat rate is also required (Charkoudian 2003). It is needless to say that to facilitate this heat exchange skin temperature needs to be greater than the environmental temperature so heat can be transferred down this thermal gradient.

2.3 Thermoregulation in Hot Conditions

Heat loss via convection, conduction and radiation is dependent on the maintenance of a temperature gradient between the skin and the environment (Nelson 1938). When the environmental temperature exceeds 35°C the gradient for heat exchange is reversed and the body will gain heat via convection, conduction, and radiation (Wendt, van Loon, and van Marken Lichtenbelt 2007). Evaporation of sweat then becomes the primary means of heat dissipation (Shibasaki, Wilson, and Crandall 2006). However, the potential for evaporative heat loss is dependent on the relative humidity. If the relative humidity is high (>60%) the evaporation of sweat is hindered resulting in a reduction in heat dissipation which can lead to development of a critical heat load (Nielsen 1996).

Exercise in hyperthermic environments ($>30^{\circ}\text{C}$) results in greater core temperatures compared to normothermic environments at the same relative exercise intensity (Cheuvront and Haymes 2001). Gonzalez-Alonso et al. (1999) reported that after 40 min of exercise at $60\% \dot{V}\text{O}_{2\text{max}}$ in hyperthermic conditions (40°C) T_{oes} increased by 3°C above resting values. Which is a greater increase than that noted in normothermic environments (20°C) exercising at a similar intensity and same duration ($\sim 1^{\circ}\text{C}$ increase from baseline at the end of exercise) (Nielsen et al. 1971). This highlights the challenge of the thermoregulatory system in an attempt to maintain homeostasis during exercise in hyperthermic conditions and the imbalance between heat gain and heat loss resulting in heat storage.

Skin temperature even at rest in hyperthermic conditions can be elevated to approximately $33\text{-}34^{\circ}\text{C}$ (Gonzalez-Alonso et al. 1999) and is further elevated to $35\text{-}38^{\circ}\text{C}$ during exercise ($60\text{-}65\% \dot{V}\text{O}_{2\text{max}}$) in hyperthermic environments ($29\text{-}40^{\circ}\text{C}$) (Rowell 1974, Bogerd et al. 2010, Cotter et al. 2001, Gonzalez-Alonso et al. 1999). Such a rise in skin temperature narrows the temperature gradient between the skin and core and thereby reduces the thermal gradient which is exacerbated in hyperthermic conditions as the thermal gradient is further reduced between the skin and the environment. The reduced heat exchange capacity would inevitably lead to further increases in core temperature which may lead to a diminished exercise performance which is discussed next.

2.4 Exercise Performance under Hot Conditions

It is well documented that exercise performance is attenuated in hyperthermic conditions ($>30^{\circ}\text{C}$) and that these conditions accelerate the onset of fatigue (Galloway and Maughan 1997). However, high intensity short-term exercise has been shown to be facilitated under conditions which increase body temperature (Navas et al. 1999, Asmussen and Boje 1945).

The following sections discuss the effects of increased body temperature at different exercise intensities.

2.4.1 High Intensity Exercise

Asmussen and Boje (1945) demonstrated that for every degree muscle temperature was increased sprint performance on a cycle ergometer increased by ~5%. However, muscle temperature appears to be limited to ~41°C (Gonzalez-Alonso, Crandall, and Johnson 2008). Muscle temperature at 41°C appears to blunt the potential advantages of an increased muscle temperature facilitating exercise performance (Gonzalez-Alonso, Crandall, and Johnson 2008). Later studies have confirmed the early finding of Asmussen and Boje (1945). For example Linnane et al. (2004) found that increasing T_{rec} temperature by 1°C, by hot water immersion (43°C) for 16 min, resulted in a significantly greater peak power output (PPO) during a 30 second sprint performance when compared to a control trial. Although muscle temperature was not measured in the Linnane et al. (2004) study it may be assumed that muscle temperature was also increased during the hot water immersion trial. An increase in muscle temperature may have provided a decrease in muscle activation and half-relaxation time. Furthermore, the increase in muscle temperature may have led to an increased maximal shortening velocity which could explain the greater PPO (Davies and Young 1983, Navas et al. 1999, Ranatunga 1982). However, it is noteworthy that there were no differences in performance in a subsequent sprint trial. Therefore, it appears that short-term anaerobic performance is facilitated by hyperthermic conditions facilitating a moderate rise in core and muscle temperature.

Additionally, Mohr et al. (2004) studied the relationship between quadricep muscle temperature and sprint performance during a football match. It was observed that an active

re-warming during half-time maintained a greater muscle temperature (2.1°C) compared to a control group (no re-warming) which resulted in a significantly greater sprint performance (2.4%) for the re-warming group. However, Davies and Young (1983) reported that an increase in muscle temperature (36.8 to 39.9°C) reduced half-relaxation time (7.7% per °C) in the tricep surae muscle but did not have an effect on maximal voluntary contraction (MVC). However, a reduction of muscle temperature by 8.4°C increased time to half-relaxation and reduced MVC. It appears that an increase in muscle and core temperature facilitates an improvement in high intensity short-term dynamic performance, however it should be noted that an upper-limit for this potential appears to exist.

2.4.2 Endurance Exercise

Conversely to anaerobic performance, prolonged exercise performance is reduced in hyperthermic conditions compared to normothermic conditions (Galloway and Maughan 1997, Marino 2002). Galloway and Maughan (1997) studied the effect of cycling to exhaustion at 70% $\dot{V}O_{2max}$ in different environmental temperatures. Time to exhaustion was 45% longer under conditions of 11°C when compared to 31°C. Performance followed an inverted U relationship as performance was longest during 11°C (93.5 ±6.2 min), and 4°C (81.4 ±9.6 min), then 21°C (81.2 ±5.7 min) and shortest during the 31°C trial (51.6 ±3.7 min). T_{rec} and skin temperatures were significantly greater in the 31°C trial compared to the 11°C trial but did not differ from the other trials. During the 31°C trial T_{rec} temperature reached 40.1°C which can be potentially critical to performance as supported by the exercise performance times observed in this study. Blood lactate was not significantly different between trials which suggests that premature fatigue during the 31°C trial was mainly due to thermoregulatory limitations as well as cardiovascular strain as HR was significantly greater during the 31°C trial compared to all other trials. Similarly, Parkin et al. (1999) examined

time to exhaustion during cycling at 70% $\dot{V}O_{2\max}$ in different ambient temperatures; 3°C, 20°C and 40°C. Exercise time was significantly longer in 3°C compared to 20°C, which in turn was longer than 40°C (85 ± 8 and 60 ± 11 and 30 ± 3 min, respectively). T_{rec} was significantly greater during exercise in 40°C when compared to 3°C and 20°C. Values for HR during 40°C was significantly greater when compared to other trials which shows evidence of cardiovascular strain. In addition, the critical T_{rec} ($\sim 39.5^\circ\text{C}$) observed in the 40°C trial may have been the predominant factor contributing to the premature fatigue.

Tatterson et al. (2000) observed the effects of a 30 min cycling time-trial in hyperthermic (32°C) and normothermic (23°C) conditions. The results demonstrated that performance was adversely affected by the hyperthermic condition. Power output was 6.5% lower during the hyperthermic condition compared to the normothermic condition. Interestingly, T_{rec} was remarkably similar between trials on completion of the time trial (39.2 ± 0.2 and $39.0 \pm 0.1^\circ\text{C}$, for hyperthermic and normothermic, respectively) which suggest that core temperature was not a limiting factor for performance. During the hyperthermic trial skin temperature was $\sim 6^\circ\text{C}$ greater than the normothermic trial as well as RPE being greater which may have affected neural and central drive to the working muscle which may explain the decrease in power output. During the last ten min of the time-trial blood lactate was significantly lower and blood pH significantly greater during the hyperthermic condition. Thus, the decrease in performance was unlikely to be mediated by cellular acidosis. During the hyperthermic trial there was an increased cardiovascular strain (greater HR values) and this may be an important determinant of perceived exertion and exercise performance. However, the exercise protocols discussed here are continuous and therefore cannot be generalised to intermittent exercise.

2.4.3 Intermittent Exercise

Drust et al. (2005) investigated the effect of 40 min intermittent cycling (15 seconds exercise and 15 seconds rest) followed by five \times 15 second sprints in hyperthermic (40°C) and normothermic (20°C) conditions. Results showed that mean power output during the five sprints were lower in the hyperthermic condition when compared to normothermic conditions. Values for core temperature on completion of exercise were significantly greater in the hyperthermic condition when compared to the normothermic condition (39.5 ± 0.2 and $38.5 \pm 0.2^\circ\text{C}$, respectively). Similar to the endurance study by Tatterson et al. (2000) blood lactate was lower during the hyperthermic condition when compared to the normothermic condition. These results suggests that the lower power output during the hyperthermic condition was not attributable to the accumulation of recognized fatigue agents such as potassium, lactate, ADP and H^+ ions. As mentioned there was no difference in core temperature in the Tatterson et al. (2000) study between trials, which maybe as a result of the lower ambient temperature (32°C) when compared to Drust et al. (2005) (40°C). The warmer ambient temperature in the Drust et al. (2005) study is one of the possible reasons for the significant difference in core temperature between hyperthermic and normothermic conditions. A greater core temperature during the hyperthermic condition may be the reason for the lower power output during the performance trial. Another possible explanation may relate to the high core temperature affecting the central drive to the working muscles thereby reducing exercise performance. The physiological limitations to exercise in the heat will be discussed in the following section.

2.5 Physiological Limitations to Exercise in the Heat

There has been extensive attention and dedicated research outlining the capacity for physical work under hyperthermic conditions in humans (Rowell 1974, Galloway and Maughan 1997,

Nybo and Nielsen 2001). Decreased exercise performance and fatigue during exercise in high ambient temperatures can be caused by multiple physiological factors (Ely et al. 2009). Previous research studying the performance limiting factors during exercise in hot conditions focused on the cardiovascular demand of simultaneously perfusing both skin, for thermoregulatory purposes, and exercising muscle for oxygen delivery (Gonzalez-Alonso, Crandall, and Johnson 2008, Crandall and Gonzalez-Alonso 2010, Rowell 1974). It is now well established that for a adequately hydrated individual, as assessed through urine osmolarity, fatigue during hyperthermic conditions is not a result of a limited perfusion of exercising muscle or baroreflex control but of high brain and core temperature (Gonzalez-Alonso, Calbet, and Nielsen 1998, Nybo and Nielsen 2001a, Nybo, Secher, and Nielsen 2002).

It has been observed that fatigue occurs at different core temperatures for untrained ($\sim 38.7^{\circ}\text{C}$) (Cheung and McLellan 1998) and trained groups ($\sim 40^{\circ}\text{C}$) (Nielsen et al. 1990, Galloway and Maughan 1997, Gonzalez-Alonso et al. 1999). Nybo and Nielsen (2001a) showed that production of force and voluntary activation percentage from working muscles (knee extensors) were significantly lower during a sustained isometric MVC under hyperthermic conditions ($T_{\text{oes}}; 40^{\circ}\text{C}$) compared to normothermic conditions ($T_{\text{oes}}; 38^{\circ}\text{C}$). However, when the femoral nerve was superimposed with electrical stimulation the resulting total force was the same in the normothermic and hyperthermic conditions. These results show that the force-generating capacity of the exercising muscle when electrically stimulated was not affected by the elevated core temperature in the hyperthermic condition. The authors concluded that the pronounced fall in voluntary contraction was as a result of reduced central drive which was attributable to the critically high core temperature. Such a high core temperature would have provided afferent information to the hypothalamus which would of

subsequently reduced force production, by decreasing muscle power output, in an attempt to attenuate any further increase in core temperature.

Research into the possible mechanisms for this reduced central drive to exercising muscles has associated fatigue with reduced arousal levels by examining changes in the electroencephalographic (EEG) signal during hyperthermic (40°C) and normothermic conditions (19°C) (Nielsen et al. 2001, Nybo and Nielsen 2001a). Participants cycled to volitional exhaustion at a fixed intensity with the α/β wave ratio measured as an index of arousal levels, with an increase in the ratio suggesting that arousal levels were reduced. Results revealed that the α/β wave ratio did increase during exercise in hyperthermic conditions which was mainly attributable to a decrease in the β activity while the α remained unchanged which reflected decreased arousal. There was a strong linear correlation ($r^2 = 0.98$) between the reduction in arousal and the increase in body temperature. These results suggest that arousal levels decrease progressively as body temperature increases rather than simply falling after core temperature reached a critical point of ~40°C. However, due to the fixed exercise intensity no graded effect of hyperthermia on performance could be established. RPE have also been observed to be greater in hyperthermic trials compared to normothermic trials in both endurance (Tatterson et al. 2000) and intermittent exercise (Drust et al. 2000) which highlights the exacerbated physiological strain associated with hyperthermia. In addition, participant mood states during exercise, measured via a Profile of Mood States questionnaire (POMS), has also been shown to be adversely affected by hyperthermic conditions which result in elevated sweat rates and subsequent fluid losses (Casa et al. 2010). These psychological changes during exercise in hyperthermic conditions may possibly exert a debilitating effect on exercise performance.

In contrast to the study design of fixed intensity exercise protocols a different picture of performance emerges during hyperthermic and normothermic conditions when a participant is able to self-pace during exercise (Tucker and Noakes 2009). Interestingly, Tucker et al. (2004) revealed that power output and muscle activation were reduced during the first 6-km of a 20-km cycling time-trial in hot (35°C) compared to cool (15°C) conditions. When pacing strategy began to differ values for, T_{rec} , RPE and HR were similar between the two conditions. In the hot condition power output was reduced as part of a possible anticipatory regulation of performance which aims to prevent excessive heat storage and increases in body temperature in order to prevent catastrophe (Tucker et al. 2004). A similar anticipatory paradigm was suggested by Marino (2004) based upon research conducted on African and Caucasian runners (Marino, Lambert and Noakes 2004). Here it has been found that body size differs between these groups (Tucker and Noakes 2009) which is an important determinant of the rate of heat storage (Dennis and Noakes 1999). In this study athletes ran for 30 min at 70% of peak treadmill speed in hot (35°C) humid (60% RH) conditions followed by a performance trial. On completion of the fixed intensity protocol, T_{rec} was identical (~38.4°C) for both the African and Caucasian runners. However, when participants commenced a self-paced run the Caucasian runners started the performance run with a significantly lower speed compared to the African runners. In addition, the African runners were able to maintain a faster mean running speed throughout the performance trial when compared to the Caucasian runners. It was concluded that the difference in pacing strategy occurred because the brain was sensitive to the rate of heat storage. Thus, the larger Caucasian runners reduced their running speed in comparison the smaller African runners to ensure excessive heat storage did not occur and to avoid lethal hyperpyrexia.

Interestingly, Morrison, Sleivert, and Cheung (2004) examined the effect of increasing core temperature on isometric knee extension while subjects were passively heated from 37.5–39.5°C and then cooled again. Participants were asked to perform a ten second MVC every 0.5°C increase in core temperature. The results show a progressive decline in force production and electrical stimulation to working muscles as core temperature was increased. Force production returned to baseline levels once core temperature was decreased with cooling. These results provide direct evidence of a centrally mediated response to a progressive rise in body temperature leading a reduced neural drive to the exercising muscle mass rather than only after a critical temperature has been reached. However, Asmussen and Boje (1945) observed that increase in muscle temperature facilitated exercise performance. It appears that direct heating of skeletal muscle improves short-term anaerobic exercise performance; albeit, increases in core temperature to a critical point appears to affect the central drive to the working muscle which results in a reduced MVC.

The mechanisms underlying central fatigue are not entirely identifiable at present but may relate to altered neuronal activity which occurs over the motor cortex area which may affect central drive to the exercising muscle mass (Nielsen et al. 2001). In addition, it has been found that during exercise in hyperthermic environments cerebral blood flow is reduced by ~18% (Nybo et al. 2002). It has been proposed that a function of cerebral blood flow is the removal of heat from the brain (Yablonskiy, Ackerman, and Raichle 2000) but has been shown that during exercise in hyperthermic conditions heat removal is impaired and is continuously stored in the brain (Nybo, Secher, and Nielsen 2002). The reduced heat removal would potentially lead to a continuous rise in brain temperature. A high hypothalamic temperature may potentially reduce the neural drive to the exercising muscle

mass in an attempt to avoid a critical temperature and avoid exhaustion or even worse heat illness.

Hyperthermia-induced fatigue appears to be, mainly, of a central origin but is not an all or none phenomenon, as explained by the critical temperature paradigm. It is better explained as a continuum, with greater core temperature resulting in greater reductions in performance (Ely et al. 2009, Cheuvront and Sawka 2001) and an anticipatory paradigm (Marino 2004, Tucker and Noakes 2009) which is concerned with cellular preservation and avoidance of catastrophe. Both latter theories state that as body temperature increases neural drive is lowered proportionally to working muscle mass. Arousal levels and mood states have also been shown to decline progressively during exercise in hyperthermic conditions which may psychologically affect participants. As a result exercise performance is reduced progressively, as oppose to the profound decline in exercise performance as stated by the critical temperature paradigm.

2.6 Cooling Methods

There are various methods of cooling that can be applied to the human body which include water-immersion (Crowley et al. 1991), water-perfused clothing (Gonzalez-Alonso et al. 1999), cold air/fans (Olschewski and Bruck 1988), and ice vests and/or ice packs (Price, Boyd, and Goosey-Tolfrey 2009). Each method has associated advantages and disadvantages. Water immersion is considered one of the best methods of cooling as heat loss to water is ~2-4 times greater than to air at the same temperature (Smith and Hanna 1975). However, the accessibility to a large water source and bath can be somewhat problematic. Water perfused suits can cover a large portion of the body, like water-immersion, however, accessibility to these type of suits is very limited. Cold air provides the

advantage of clamping skin temperature at similar levels across the body although most individuals do not have access to a facility that provides the cold temperature needed. Ice vests, as well as ice packs, are very practical to the field setting as ice vests are commercially available and accessible and can be applied prior to exercise performance or mid-event, however, ice vests and packs do not cover large regions of the body. Thus, the cooling power of this method is not as substantial as other methods (Bogerd et al. 2010). Methods such as water immersion have the most profound cooling power (Wilson et al. 2007).

The primary aim of pre-cooling is to reduce body temperature prior to exercise which increases the margin for metabolic heat production thereby increasing the time to which upper limits of core temperature are reached (Marino 2002). Research within this area has vast methodological approaches in relation to cooling techniques (modality and duration) and exercise protocols which makes it difficult to make comparisons between studies. For ease of comparison between studies the discussion of previous research has been separated according to exercise protocol.

2.6.1 High Intensity Exercise

Cooling prior to high intensity performance in the heat would presumably result in a decrease in power output due to the association that lower muscle temperature is directly linked to lower power output (Asmussen and Boje 1945, Davies and Young 1983). Crowley et al. (1991) demonstrated that pre-cooling of the legs by water immersion (11.5-12.2°C) for 30 min prior to a 30-second Wingate test resulted in peak power and average power output to decline by 30% and 26%, respectively, compared to a control trial of no pre-cooling. Although muscle temperature was not measured during this study it is likely that lower muscle temperature was the cause of the reduced power output. In addition, Sleivert et al.

(2001) found that torso and thigh cooling (combination of ice vest, cold air, and water-perfused cuffs on thighs) without warm-up, prior to a 45-second sprint cycling test in hyperthermic conditions (33°C). Values for core (mean of T_{rec} and T_{oes}) temperature were 36.8 ± 0.4 and $37.3 \pm 0.3^\circ\text{C}$ for cooling and control trial, respectively. Muscle temperature was significantly lower following torso and thigh pre-cooling compared to the control trial (~ 35 and $\sim 37^\circ\text{C}$, for pre-cooling and control, respectively). Results show that following a cooling period there was a significant reduction in peak (7.7%) and mean power output (7.6%) when compared to a control trial. It was postulated that a decrease in muscle temperature was the likely reason for the decline in performance in comparison to the control trial. Torso-only pre-cooling did not reduce mean power or peak power output, either with or without warm-up. Sleivert et al. (2001) provides further evidence of the attenuated muscular force for skeletal muscles when directly cooled prior to sprint performance.

Marsh and Sleivert (1999) utilised 30 min of torso-only water immersion (legs removed from the water) followed by a ten min warm-up ($60\% \dot{V}O_{2\text{max}}$) prior to a 70-second sprint cycle in the heat (29°C). Participants sat in a water bath with an initial temperature of 18°C which was reduced to $12\text{--}14^\circ\text{C}$ after 5 min reducing T_{rec} from 37.2 to 36.9°C which was significantly lower than the control trial (37.1°C). Values for RPE at the end of the warm-up were significantly lower in the cooling trial compared to the control trial. In addition, T_{rec} was significantly lower at the end of the 10 min warm-up in the cooling trial compared to the control trial and was significantly lower at the end of the performance test (~ 36.6 and $\sim 37.8^\circ\text{C}$, for cooling and control, respectively) as well as significantly improving power output by 3.3%. The authors suggested that the increase in performance was attributable to the cold induced cutaneous vasoconstriction which may have led to an increase in central blood volume which would have provided additional muscle blood flow allowing for

improved oxygen delivery and waste removal. However, skeletal muscle blood flow has been shown to not be reduced during exercise in the heat (Nielsen, Savard, and Richter 1990) so is therefore questionable whether the possible increase in muscle blood flow was the mechanism responsible for the ergogenic effect of pre-cooling. It could be speculated that although T_{rec} was reduced due to cooling, muscle temperature was maintained and therefore did not limit performance and was facilitated subsequently. Therefore, it appears to that pre-cooling applied direct to the potential working muscle is debilitating to high intensity exercise. Whereas pre-cooling procedures that lower core temperature and potentially maintain muscle temperature appear successful in improving high intensity performance.

2.6.2 Endurance Exercise

In contrast to the high intensity exercise studies there is substantial evidence to suggest that pre-cooling improves endurance exercise in hyperthermic and normothermic conditions (Marino 2002). Olschewski and Bruck (1988) examined a double pre-cooling exposure (2×15 min) on endurance exercise performed on a cycle ergometer. Participants sat in a room with an ambient temperature between 5-10°C. An intermittent re-warming period was utilised in an attempt to blunt any thermogenesis. After pre-cooling participants cycled for 16 min of sub-maximal exercise at an ambient temperature of 18°C and then immediately performed the performance test which was to cycle at 80% $\dot{V}O_{2peak}$ to exhaustion. Pre-cooling reduced T_{oes} and skin temperature by 0.2 and 4.0°C, respectively, with HR values being significantly lower throughout exercise compared to the control trial. Furthermore, time to exhaustion was 12% longer with pre-cooling compared to the control trial (18.5 ± 2.5 and 20.8 ± 2.3 min, respectively). The improved time to exhaustion with pre-cooling may be as a result of the small, but significant, lower starting T_{oes} therefore increasing heat storage

capacity in addition to a reduced cardiovascular strain. Although RPE was not measured in the study this may have been affected positively following the cooling period.

Booth, Marino, and Ward (1997) conducted a self-paced 30 min running time trial in hot humid conditions (32°C, 60% RH) preceded by cooling. Pre-cooling (up to 60 min) involved cold-water immersion up to the neck level. Water temperature was gradually reduced from 28-29°C to 23-24°C in an attempt to minimize subject discomfort and thermogenesis. T_{rec} and mean skin temperature was reduced by 0.7°C and 5.9°C, respectively, and remained decreased throughout exercise up to 20 and 25 min, respectively. Pre-cooling significantly reduced HR at rest and at five and ten min of exercise. Run distance increased significantly by 304 meters (4%) following the cooling period compare to the control. The reduced T_{rec} and skin temperature in addition to the reduced cardiovascular strain may have affected the central drive to the working muscle mass and in addition may have affected arousal levels positively and thereby increasing exercise performance. Olschewski and Bruck (1988) reported a 0.2°C reduction in T_{oes} following the cooling period which is not as substantial as the reduction of 0.7°C of Booth, Marino and Ward's study. Although different sites of core temperature were measured the difference in core temperature reductions reported may be due the different methods of pre-cooling. In addition, Olschewski and Bruck (1988) only pre-cooled their participants for a total time of 30 min whereas Booth, Marino, and Ward (1997) study pre-cooled for up to 60 min.

Although effective in reducing body temperature water immersion may not be available to an individual immediately prior to performance and so therefore a more practical modality of cooling may need to be utilised. Peiffer et al. (2008) examined the effects of cooling mid event between 2 × 25 min of constant of pace cycling (65% $\dot{V}O_{2\text{max}}$) with a 15 min break

between bouts performed in hyperthermic conditions (35°C, 40% RH). Following each bout of 25 min of exercise a 4-km time trial was completed. The 15 min recovery time included five min of cooling via water immersion at 14°C to mid-sternal level. At the end of the 15 min recovery time T_{rec} was reduced by $0.4 \pm 0.4^{\circ}\text{C}$ in the cooling trial compared to the control trial but was still elevated above baseline. In addition, RPE was significantly lower after cooling at the end of the second bout of 25 min of exercise compared to control (14.3 and 16.8, for cooling and control, respectively). The mean performance time of the second 4-km time trial was 18 seconds less following the cooling period compared to the control trial. The significant improvement in performance following cooling could be attributed to both the lower T_{rec} and RPE which may have significantly improved central drive to the working muscle as well the participants' psychological feeling of arousal, readiness to exercise and/or mood states. The cooling methods utilised within this study are more applicable to field settings as it is more practical and shorter in duration.

Arngrimsson et al. (2004) utilised ice vests for pre-cooling during which participants performed a 38 minute warm up prior to a 5-km time trial run. Both the warm-up and time trial were performed in hyperthermic conditions (32°C, 50% RH). T_{rec} after the warm-up was significantly lower in the pre-cooling trial compared to the control trial (38.0 ± 0.4 and $38.2 \pm 0.4^{\circ}\text{C}$, respectively) but remained elevated above baseline. In addition, the cooling vest significantly reduced thermoregulatory (thermal discomfort) and cardiovascular strain (lower HR) during the warm-up compared to the control trial. Performance in the 5-km run was significantly improved by 1.1% in the pre-cooling trial compared to the control trial (1147 ± 130 and 1134 ± 132 seconds, respectively). T_{rec} , in the pre-cooling trial, remained significantly lower compared to the control during the time trial until 3.2 km. The authors concluded that wearing an ice vest during an active warm-up was effective in improving

performance by attenuating the rise in core temperature as well as reducing thermal and cardiovascular strain. The adaptations provided by pre-cooling result in a larger margin for metabolic heat production and enable participants to maintain a faster pace during exercise performance.

Although research in the area of pre-cooling prior to exercise in hyperthermic conditions is vast and shows an ergogenic effect, many studies have only speculated a reason for the notable improvement in performance. It has been suggested that pre-cooling may blunt a rise in core temperature by causing initial cutaneous vasoconstriction (Wilson et al. 2007) which could potentially abate the increase in cutaneous blood flow during exercise (Price, Boyd, Goosey-Tolfrey 2009). Subsequent exercise may cause core temperature to rise which will lead to an increase in skin blood flow. As a result the blood perfusing the cooled skin would subsequently be cooled and attenuate a rise in core temperature on return (Price, Boyd, Goosey-Tolfrey 2009).

Gonzalez-Alonso et al. (1999) measured forearm skin blood flow, via laser-Doppler, during exercise in the heat (40°C) preceded by cooling via water-immersion. Participants were required to cycle to volitional exhaustion at 60% $\dot{V}O_{2max}$. It was observed that T_{oes} was reduced by 1.5°C following pre-cooling compared to the control trial but were similar at exhaustion (~40.2°C). It was also observed that forearm skin blood flow during exercise in the pre-cooling group was not significantly different from the control trial. Time to exhaustion was significantly longer in the pre-cooling trial compared to the control trial (63 ±3 and 46 ±3 min, respectively). The study conducted by Gonzalez-Alonso et al. (1999) does not support the mechanism proposed by Price, Boyd, and Goosey-Tolfrey (2009). However, only forearm skin blood flow was measured during this study and therefore cannot draw

conclusions in relation to local vasoconstriction from one measured site or from cooled versus non-cooled sites.

Similarly, Bogerd et al. (2010) measured skin blood flow change, via laser-Doppler, from pre to post cooling (ice vest for 45 min) and control trials in normothermic conditions (25°C).

Following this, participants cycled to volitional exhaustion at 65% $\text{VO}_{2\text{peak}}$ in hot humid conditions (29°C, 80% RH). It was observed that skin blood flow was significantly reduced at the end of pre-cooling on six sites (back, chest, shoulder, thigh, calf, and finger) compared to the control trial. In addition, mean skin temperature decreased from 33.5 ± 0.3 to $30.6 \pm 0.6^\circ\text{C}$ during pre-cooling. Despite this lower skin temperature in the pre-cooling trial T_{rec} was not significantly reduced compared to the control trial. This shows that the ice vest lowered skin temperature likely causing cutaneous vasoconstriction shown through a reduction in skin blood flow. It is possible that the reason why there was only a small non-significant reduction in T_{rec} was as a result of vasoconstriction of the cutaneous vasculature resulting in a lower perfusion of the cooled skin which prevented the blood being cooled which subsequently may have cooled the core.

Time to exhaustion was five min longer following the cooling trial compared to the control (41.46 ± 07.02 and 36.44 ± 09.20 min, respectively). Skin temperature at exhaustion was similar for pre-cooling and control trial (37.2 ± 0.4 and $37.5 \pm 0.4^\circ\text{C}$, respectively), in addition to T_{rec} being identical in both trials at exhaustion ($39.1 \pm 0.5^\circ\text{C}$). Although T_{rec} was identical at exhaustion it was lower throughout exercise during the pre-cooling trial with the control trial exhausting first followed by the pre-cooling trial. These results show that ice vest pre-cooling was effective at improving endurance performance compared to the control trial. Despite this, Bogerd et al. (2010) did not measure skin blood flow during exercise and

Gonzalez-Alonso et al. (1999) only measured one skin site with future research needing to expand on the latter studies findings.

2.6.3 Intermittent Exercise

Much of the research dedicated to pre-cooling is continuous in nature which may not be representative of a field setting due to the difference in activity patterns represented in many sports. Castle et al. (2007) studied the effect of 20 min of three different cooling methods (ice vests, water immersion or ice packs to thighs) and a control trial prior to intermittent performance in the heat (34°C, 52% RH). Participants were required to complete 20 × two min bouts of exercise on a cycle ergometer which included; ten seconds of passive rest, five seconds of maximal sprint (against 7.5% of body mass) and 105 seconds of active recovery. All pre-cooling trials reduced skin temperature (~30, ~21, and ~29°C for vest, water and ice packs, respectively) compared to the control trial (~32°C). In addition, T_{rec} was lowered by a small non-significant amount in all pre-cooling trials (37.3, 37.3, and 37.1°C, for vest, water and ice packs, respectively) compared to the control trial (37.5°C).

Castle et al. (2007) observed peak power output (PPO) for the control trial to progressively decline from sprint one whilst the pre-cooling trials managed to maintain a relatively stable PPO throughout the trial. Skin temperature at completion of the protocol was significantly greater in the control, ice packs, and ice vest trial (~36°C) compared to the water immersion trial (~34°C). However, T_{rec} at completion was approximately 38.8, 38.3, 38.6 and 38.9°C for vest, water, ice packs and control, respectively. All trials increased PPO in the final sprint compared to the penultimate sprint. It can therefore be highlighted that peripheral fatigue was not the cause of the decline in PPO as there was an increase in PPO in the final sprint. It seems more likely that the participants, at least at a subconscious level, appeared to pace

themselves based on their core and skin temperature providing afferent information to the hypothalamus. Interestingly, the greatest increase in PPO in the final sprint was observed in the ice vest trial despite providing the smallest dose of pre-cooling in relation to reductions in skin and core temperature. The authors suggested that this may be attributable due to the ice vest providing less sensory information to the brain (Noakes 2004). Thus, resulting in a pacing strategy that was too conservative which was recalculated towards the end of the protocol resulting in a greater PPO in the final sprint.

Conversely to Castle et al. (2007), Duffield et al. (2003) observed that pre-cooling utilising an ice vest did not result in improved performance during intermittent exercise compared to a control trial. Participants were required to complete 80 min of an intermittent sprint cycling protocol in hot conditions (30°C, 60% RH) with or without pre-cooling. The protocol was designed to simulate team sports by using sprints and active recovery with quarter and half-time breaks. Pre-cooling was applied five min prior to the start of exercise, with two \times five min periods and one \times ten min period during quarter and half-time breaks, respectively. Rectal and skin temperature did not significantly differ between trials at the start of each quarter. T_{rec} increased similarly throughout the test between trials (1.2 and 1.4°C, for pre-cooling and control, respectively). Thermal comfort and RPE across the duration of the test did not significantly differ between trials. In addition, POMS data showed that no significant differences were noted between trials for all mood states measured. Total mean power (W) throughout the test was not significantly different between cooling and control trials. It is highly likely that the cooling intervals used within this study were not sufficient in duration to elicit any significant change in core or skin temperature, thermal comfort, RPE or mood states which may be the reason no significant difference was observed in exercise performance between trials. Although pre-cooling was only applied for five and ten min at

any one time in the Duffield et al. (2003) study to represent teams sports, there would be many other situations where an individual would be able to apply pre-cooling for longer periods of time prior to exercise.

A more recent study that applied ice vests for a longer duration was conducted by Price, Boyd, and Goosey-Tolfrey (2009). Pre-cooling, as well as pre and mid-exercise cooling, were utilised prior to two bouts of 45 min of intermittent exercise with a 15 min recovery time during hyperthermic conditions (31°C, 64% RH). A control trial of no cooling was also included within the study. The ice vest was applied 20 min prior to exercise with mid-event cooling lasting 15 min. Mean skin temperature after initial cooling was significantly lower in both cooling trials (31.3 ± 0.9 and $31.5 \pm 1.2^\circ\text{C}$, for pre, pre and mid cooling, respectively) in comparison to the control trial ($33.5 \pm 0.4^\circ\text{C}$). However, T_{rec} was not significantly reduced at the end of cooling in comparison to the control trial in either of the two cooling trials (0.2 ± 0.1 and $0.1 \pm 0.2^\circ\text{C}$, for pre, pre and mid, respectively). The change in T_{rec} during exercise was significantly greater in the control trial compared to the two cooling trials from 35 min until the end of the second exercise bout. At the end of the half-time period, mean skin temperature was significantly lower during pre and mid-event cooling ($33.4 \pm 0.7^\circ\text{C}$) compared to pre-cooling ($34.5 \pm 0.3^\circ\text{C}$) and control trial ($34.7 \pm 0.4^\circ\text{C}$). These results seem to contrast Duffield et al. (2003) as values for T_{rec} and skin temperature were not significantly different between cooling and control trials. Again, this may be due to the Duffield et al. (2003) study not cooling for a sufficient time to elicit any significant affects in T_{rec} .

Price, Boyd, and Goosey-Tolfrey (2009) suggested that the blunted rise in core temperature following a cooling period, as aforementioned, may be as a result of the ice vest causing local vasoconstriction. Therefore, as body temperature increased during exercise, the demand for

skin blood flow would increase and as a result the blood perfusing the skin would subsequently be cooled which would attenuate a rise in core temperature on return via venous circulation. However, despite a potential mechanism suggested of how pre-cooling lowers core temperature, skin blood flow was not measured in the study.

2.7 Summary

Exercise disturbs the homeostatic state of the human body due to the inefficiency of exercise causing an increase in heat production within the body which is exacerbated when exercise is undertaken in hyperthermic conditions. The hypothalamus attempts to dissipate heat from the core to the skin via an increase in skin blood flow to approximately $8 \text{ L} \cdot \text{min}^{-1}$ (Charkoudian 2003) in an attempt to abate the rise in core temperature which is facilitated by an increased sweat rate. Price, Boyd and Goosey-Tolfrey (2009) speculated that pre-cooling may cause local vasoconstriction which would result in skin blood flow to be blunted during subsequent exercise performance. The increase in core temperature during subsequent exercise increases the demand for skin blood flow with the blood flowing through the cutaneous circulation being cooled more so in comparison to non pre-cooled individuals. The mechanism proposed by Price, Boyd and Goosey-Tolfrey (2009) could possibly be the reason by which pre-cooling attenuates the rise in core temperature during exercise in the heat. Despite the possible speculative reasons suggested by various authors, Bogerd et al. (2010) and Gonzalez-Alonso et al. (1999) have provided data for skin blood flow changes. It was shown firstly that skin blood flow was reduced as a result of pre-cooling (Bogerd et al. 2010) but not measured during exercise and secondly skin blood flow during exercise in the heat preceded by a cooling period was not significantly lower compared to a control trial (Gonzalez-Alonso et al. 1999) although only one site was measured. Therefore, future research should measure skin blood flow at various locations during pre-cooling and exercise

performance in the heat in an attempt to identify a possible mechanism of how pre-cooling reduces core temperature. This would attempt to address the mechanism suggested by Price, Boyd, and Goosey-Tolfrey (2009). In addition, a seated rest in the heat protocol preceded with a cooling period should be carried out to observe the contribution of cooling without exercise metabolic heat production. Finally, psychological assessments on participants should be carried out to assess their thermal strain, readiness to exercise and mood states to observe any differences between cooling and control trials to help explain any improvements in exercise performance.

2.8 Aim and Hypotheses

2.8.1 Aim

The aim of this study was to determine whether pre-cooling, with an ice vest, reduces skin blood flow compared to a no cooling control and subsequently improve exercise performance in the heat. A secondary aim was to determine the effects of ice vest pre-cooling on seated rest and heat exposure to observe the responses of cooling compared to a control without exercise metabolic heat production.

2.8.2 Hypotheses

Ice vest pre-cooling will significantly reduce skin blood flow and mean skin temperature during cooling and will lead to greater improvements in a subsequent performance trial compared to a control trial. Core temperature during exercise will be significantly lower during exercise compared to a control trial.

Ice vest pre-cooling will significantly reduce skin blood flow during seated rest in the heat compared to a control trial. Core temperature during seated rest in the heat will be significantly lower during exercise compared to a control trial.

3.0 Methods

3.1 Participants

Eight male participants volunteered to take part in the study. The mean (\pm SD) age, height, weight and maximal oxygen uptake ($\dot{V}O_{2\max}$) were; 24.5 ± 5.0 years, 178.7 ± 2.6 cm, 77.5 ± 13.7 kg and 43.4 ± 8.6 ml.min⁻¹kg⁻¹, respectively. The study was approved by Coventry University Faculty of Health and Life Sciences Ethics Committee (Appendix 1). Participants were provided with a participant information sheet (Appendix 2) which outlined all the demands and risks involved in the study. Written informed consent was obtained from each participant prior to participation in the study. All participants were actively taking part in recreational exercise for ~4 hours per week which included football, cycling, running or weightlifting. In addition, all participants were healthy and free from any illness and chronic disease as assessed via a health screen questionnaire which was completed prior to each testing session (Appendix 3). Each participant was asked to refrain from alcohol and exercise for 24 hours prior to each trial and not consume any caffeine two hours prior to each trial.

3.2 Preliminary Testing

On the first visit to the laboratory each participant completed an incremental exercise protocol on a cycle ergometer (Ergomedic 874E, Monark, Sweden) to determine $\dot{V}O_{2\max}$. The test required the participant to cycle at an initial power output of 70 W with increases of 35 W every three min. Participants were asked to maintain a cadence of 70 rev.min⁻¹ until volitional exhaustion criteria were reached (i.e. drop in cadence by 5 rev.min⁻¹). Expired gas was sampled and analysed via a breath-by-breath gas analyser (Metamax 3B, Cortex Biophysik, Germany). The analyser was attached to the participant via a face mask and sample line. The breath-by-breath analyser was calibrated prior to every exercise test for

atmospheric pressure (mmHg) (Barometer; F. Darton & Co. Ltd, Watford) with a known volume of gas (3 litre calibration syringe; Hans Rudolph, USA) and a known concentration of oxygen and carbon dioxide (15% and 5%; O₂ and CO₂ respectively, Nitrogen balance, BOC Gases). HR was measured using a Polar Heart Rate Monitor (Polar S610i, Polar Electro LTD, Finland). Values were recorded in the final 15 seconds of each workload and at exhaustion. Ratings of perceived exertion (RPE) were also measured during the final 15 seconds of each stage and at exhaustion using the Borg scale (Borg 1973, Appendix 4). Verbal encouragement was given throughout the test. Fingertip arterialized capillary blood samples were collected (20 µL capillary tubes; Analox, LMS, UK) after five min of seated rest and one and five min post exercise and were analysed for blood lactate ([BLa]) using a Biosen C_Line analyser (EKF Diagnostic, Germany). All tests took place in an air conditioned laboratory (19.2 ±0.2°C).

3.3 Experimental Trials

Participants subsequently visited the laboratory on five occasions which consisted of; one familiarisation exercise trial, two exercise trials and two seated rest trials. Both the exercise and seated rest trials involved either: an ice vest pre-cooling trial (COOL) or a non-cooling control trial (CON). Each trial was carried out at the same time of day to negate circadian variation (Hill *et al.* 1993) (and no less than a week apart), to ensure there was adequate rest between trials and to prevent acute effects of heat acclimation (Price, Boyd, and Goosey-Tolfrey 2009). The order of testing was counter-balanced. All experimental trials were performed at 35.4 ±0.4°C with a 26.3 ±4.1% relative humidity. The familiarisation trial consisted of participants undertaking the full experimental protocol in the hot condition.

3.3.1 Protocol

Participants were required to wear a t-shirt, shorts, socks and training shoes. For each trial, participants were prepped in an air conditioned laboratory maintained at $19.7 \pm 0.4^{\circ}\text{C}$. Nude body weight was measured prior to testing using balance scales (Seca 888, UK). At the start of each experimental trial the participants were required to rest for ten min in normothermic conditions. After this participants were required, dependent upon trial, to wear an ice vest (Arctic Heat, Burleigh Heads, Australia) for 20 min while remaining seated. Those not undertaking the ice vest trial remained seated for a further 20 min. Participants were then required to remove the ice vest as appropriate and move in to the heat chamber. Once in the heat chamber, the participant sat and rested for a further five min while breathing into a gas analyser. The participant then either continued with a period of seated rest during the heat trial or performed an exercise trial. The exercise protocol consisted of 45 min of intermittent exercise including a range of intensities between 40-100% $\dot{V}\text{O}_{2\text{max}}$ obtained from the preliminary trial (Figure 3.1). The protocol, adapted from Price, Moss, and Rance (2003) involved nine \times five min bouts of exercise consisting of; 2 min 30 seconds at 40% $\dot{V}\text{O}_{2\text{max}}$, 1 min 30 seconds at 60% $\dot{V}\text{O}_{2\text{max}}$, 30 seconds at 100% $\dot{V}\text{O}_{2\text{max}}$, and 30 seconds of unloaded cycling at a cadence of $70 \text{ rev}\cdot\text{min}^{-1}$. After completion of the exercise, three min rest was allowed for physiological and perceptual measures to be taken. Following this, participants were required to complete a three kilometre performance trial (PT) at a resistance set at 60% $\dot{V}\text{O}_{2\text{max}}$ with the instruction to complete the distance as fast as possible. Cadence was therefore freely selected by the participant and recorded at each half-kilometre. Participants completing a seated rest trial finished the experimental trial after 45 min and were not required to complete the PT. The seated rest trials were carried out to provide data on the physiological effects of pre-cooling without an exercise stimulus to highlight the differences in physiological and psychological responses between exercise and seated rest in the heat.

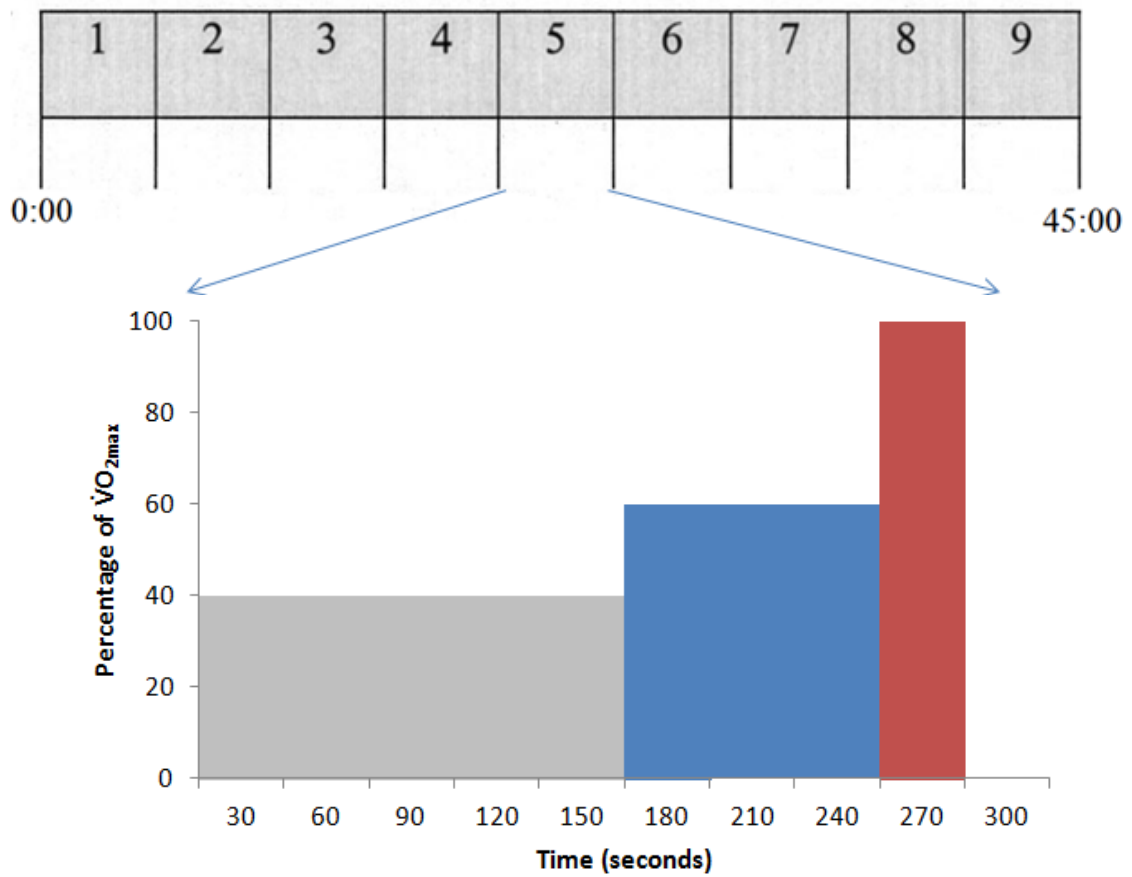


Figure 3.1. Schematic diagram of one five min block of the exercise protocol out of nine in total.

3.3.2 Physiological Measures

Oxygen consumption ($\dot{V}O_2$) was continually monitored during the experimental trials which was averaged over 10 seconds using the Metamax at 6, 16, 31 and 41 min of exercise which were recorded during the 40% $\dot{V}O_{2max}$ intensity bouts. Measurements of $\dot{V}O_2$ was also recorded in the final kilometre of the PT. Due to equipment failure expired gas samples were alternatively collected via Douglas bags for one min during the respective periods where appropriate (6, 16, 31 and 41 min of exercise and final kilometre of the PT). Measurement of HR was continually monitored during all trials and was recorded at five min intervals for exercise and seated rest trials. Fingertip arterialised capillary blood samples were obtained for the analysis of haemoglobin ([Hb]), haematocrit (Hct) and [BLa]. Samples of [Hb] and Hct were obtained at baseline and immediately post PT. Samples for [BLa] were also taken at baseline, at 5, 15, 30 and 45 min of exercise and post PT. Values for [Hb] were analysed in duplicates using Hemocue cuvettes (Hemocue AB, Angelholm, Germany) and was measured using a Hemocue photometer (Hemocue Ltd., UK). Hct was measured in triplicate using 75 μ L heparinised capillary tubes which were plugged at one end (Hawksley & Sons Ltd, England) and centrifuged at 3000 rpm for 15 min (Haematokrit 210, Hettich, Germany) and read using a Hawksley Micro Haematocrit reader. Changes in plasma volume were calculated using the formula of Dill and Costill (1974). See Figure 3.2 for schematic of measurements taken during exercise.

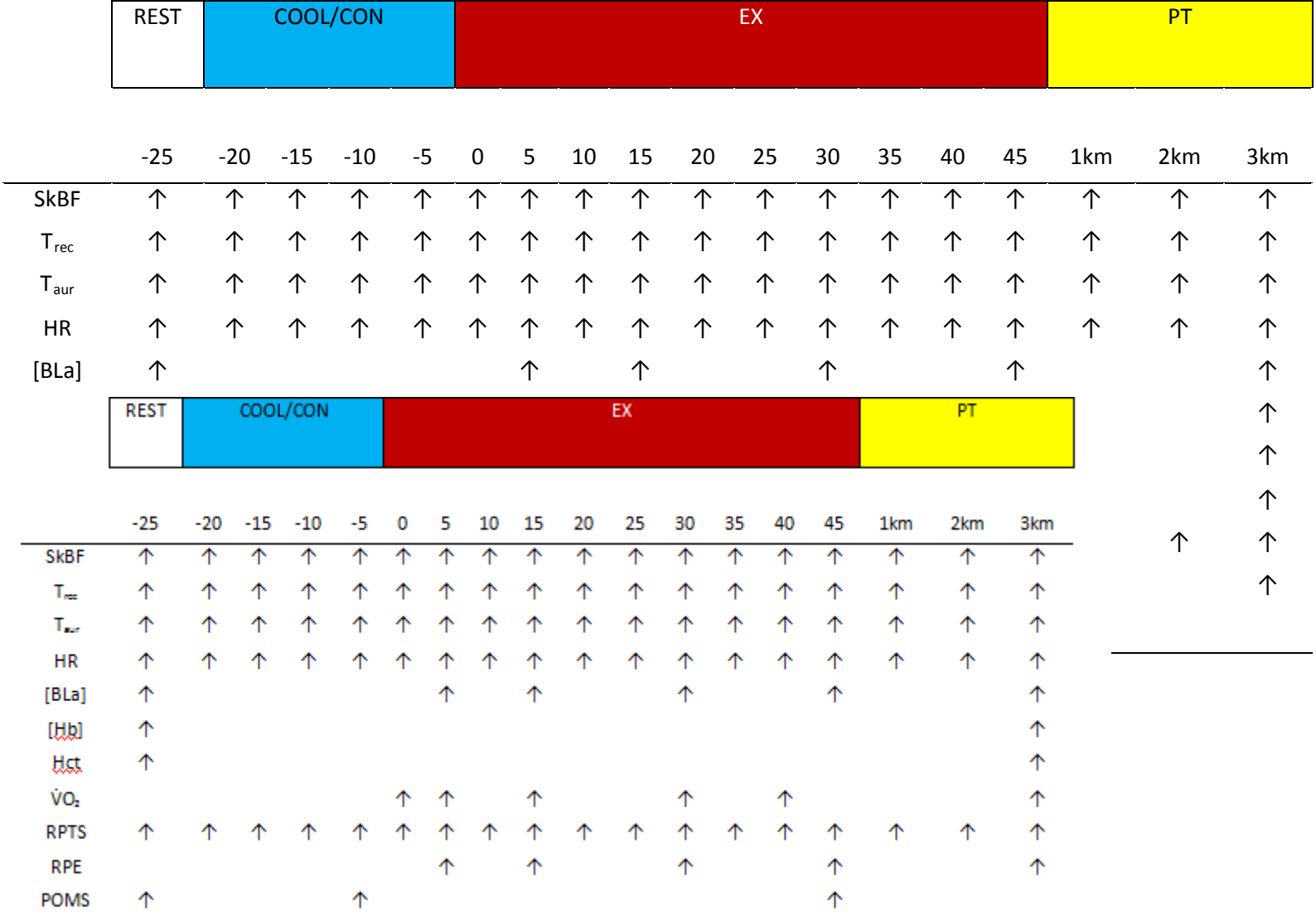


Figure 3.2. Schematic diagram of measurements at rest (REST), during cooling (COOL/CON), exercise protocol (EX), and performance trial (PT).

Core temperature was recorded from T_{rec} and T_{aur} temperature thermistors. T_{rec} was measured by a rectal thermistor inserted 10 cm beyond the anal sphincter. T_{aur} was measured by inserting an aural thermistor into the ear and insulating the external ear with cotton wool. Skin temperature was measured via skin thermistors which were attached to the participant at five sites using adhesive tape (calf; upper third lateral aspect of the leg, thigh; midpoint anterior aspect of the left femur, bicep; midpoint anterior aspect of the left bicep, chest; midpoint of the left pectoral muscle, and back; medial aspect of the left scapula). Values for T_{rec} , T_{aur} , and skin temperature were recorded via a Squirrel 1000 series data logger (Grant Instruments, Cambridge, England) every five min. Mean skin temperature (T_{ms}) was calculated according to the method of Ramanathan (1964) as follows:

$$T_{\text{ms}} = 0.3 (T^{\circ}_{\text{bicep}} + T^{\circ}_{\text{back}}) + 0.2 (T^{\circ}_{\text{thigh}} + T^{\circ}_{\text{calf}})$$

Where: T°_{bicep} = Temperature of the bicep

T°_{back} = Temperature of the back

T°_{thigh} = Temperature of the thigh

T°_{calf} = Temperature of the calf

Skin blood flow (SkBF) was continuously monitored throughout the experimental trial using laser-Doppler probes (MoorLAB, Moor Instruments Ltd., Devon, England) attached to four skin sites (thigh; midpoint anterior aspect of the right femur, bicep; midpoint anterior aspect of the right bicep, chest; midpoint of the right pectoral muscle, and back; medial aspect of the right scapula) using adhesive tape (Figure 3.3).

This image has been removed for data protection reasons.
The unabridged version of the thesis can be viewed at the
Lanchester Library, Coventry University.

Figure 3.3. Participant completing a seated rest protocol in the heat chamber. Bicep and chest laser-Doppler probes and skin thermistors are visibly depicted.

3.3.3 Perceptual Measures

A shortened Profile of Mood States (POMS, Appendix 5) was developed as the length of the original POMS questionnaire (McNair, Lorr, and Droppleman 1971) meant it was not suited to administration during exercise. The original POMS (Appendix 6) questionnaire includes 65 adjectives categorised into six subscales; Anger, Fatigue, Vigour, Depression, Tension and Confusion. Each adjective is ranked on a Likert-type scale from 0 (“Not at all”) to 4 (“Extremely”) with each subscale based on the response to the 65 adjectives. The shortened version used the same six subscales as the original with 3-4 of the original associated subscale words listed as prompts.

In order to validate the shortened version of the POMS an initial matched group of participants ($n = 15$) in relation to age and gender were asked to report how they felt under a

non-competitive situation in relation to each of the six scales on the shortened and original scales. Validation of the modified version required participants to complete the shortened POMS and original POMS at the same time. Correlation analysis for the two questionnaires showed a high correlation ($r = 0.74-0.90$) for each subscale between the original and shortened POMS (anger $r = 0.90$, fatigue $r = 0.83$, vigour $r = 0.85$, depression $r = 0.90$, tension $r = 0.86$, confusion $r = 0.74$) and were not significantly different from one another ($P > 0.05$) (Appendix 7). This provided validity for the shortened POMS which provided a quicker alternative to the original POMS questionnaire.

The modified POMS was administered at baseline, end of cooling and immediately after exercise (45 min) and upon completion of the seated rest during the heat (45 min). Perceived readiness to exercise (RTE) (Nurmekivi *et al.* 2001, Appendix 8) was administered at the same time as the POMS. Each participants' rating of perceived thermal sensation (RPTS) (Young *et al.* 1987, Appendix 9) was recorded at the same time as T_{rec} , T_{aur} and skin temperature. RPE was taken at the same time as the blood samples during exercise.

3.4 Statistical Analysis

All data was presented as mean (\pm SD). A two-way analysis of analysis of variance (ANOVA) with repeated measures on both factors (trial \times time) was used to determine statistical differences. This was carried out using SPSS software, version 17.0 (SPSS Inc., Chicago, Ill.). Statistical significance was set at $P < 0.05$. Where significance was achieved, Tukey's post hoc analysis was undertaken by using the following formula:

$$HSD = q_{(k, dfE)} \frac{\sqrt{MSE}}{N}$$

Where: q = value from the studentized range distribution

k = number of groups

df_e = degrees of freedom

MSE = mean square error

n = number of participants

A T-test was performed (Microsoft Excel 2007) to compare the performance results of the PT between COOL and CON. A Pearson Rank correlation was also carried out (Microsoft Excel 2007) out to validate the shortened POMS questionnaire. Correlation analysis and a repeated measures ANOVA was carried out to provide reliability of ice vest pre-cooling procedures on T_{ms} , and T_{back} and RPTS. In addition, a correlation analysis was carried out to analyse the relationship between HR and RPE utilising SPSS.

4.0 Results

4.1 Seated Rest Trials

4.1.1 Physiological Responses

Values for HR and $\dot{V}O_2$ did not differ between trials or over time for the seated rest trial (Table 4.1). There was a significant main effect for time for HR ($P=0.05$) but not $\dot{V}O_2$ ($P=0.63$). No significant interactions were observed for [BLa] ($P=0.71$), [Hb] ($P=0.20$) and Hct ($P=0.49$) between trials (Table 4.1).

The amount of total body mass lost between COOL (0.2 ± 0.0 kg) and CON (0.1 ± 0.1 kg) was not significantly different ($P=0.50$) as a consequence of a similar sweat rate ($L \cdot h^{-1}$) between trials (0.13 ± 0.18 and $0.27 \pm 0.15 L \cdot h^{-1}$, for COOL and CON, respectively) ($P=0.57$). There was no significant difference in percentage change of plasma volume between COOL (1.51 ± 6.12) and CON (-3.10 ± 6.58) ($P=0.17$).

Table 4.1. Mean \pm SD for HR, $\dot{V}O_2$, [BLa], [Hb] and Hct at baseline (-25 min), end of cooling (-5 min) and end of seated rest in the heat (45 min).

		-25 min	-5 min	45 min
HR (beats.min ⁻¹)	COOL	72 \pm 5	65 \pm 6	71 \pm 6
	CON	74 \pm 9	72 \pm 8	71 \pm 6
$\dot{V}O_2$ (L.min ⁻¹)	COOL	0.30 \pm 0.09	-	0.32 \pm 0.06
	CON	0.29 \pm 0.07	-	0.29 \pm 0.04
[BLa] mmol.l ⁻¹	COOL	1.00 \pm 0.36	-	0.79 \pm 0.21
	CON	1.01 \pm 0.22	-	0.79 \pm 0.13
[Hb] d.L ⁻¹	COOL	158 \pm 9	-	159 \pm 10
	CON	149 \pm 11	-	155 \pm 13
Hct %	COOL	44 \pm 3	-	45 \pm 3
	CON	44 \pm 2	-	44 \pm 3

SkBF for the thigh, bicep, chest and back during cooling and seated rest in the heat in both COOL and CON are shown in Figures 4.1 to 4.4, respectively. All SkBF sites followed the same trend. No significant interactions for SkBF were observed at any site between COOL and CON at any time point. However, there was a significant main effect for time for the thigh ($P<0.001$), bicep ($P<0.001$), chest ($P<0.001$) and back ($P<0.001$).

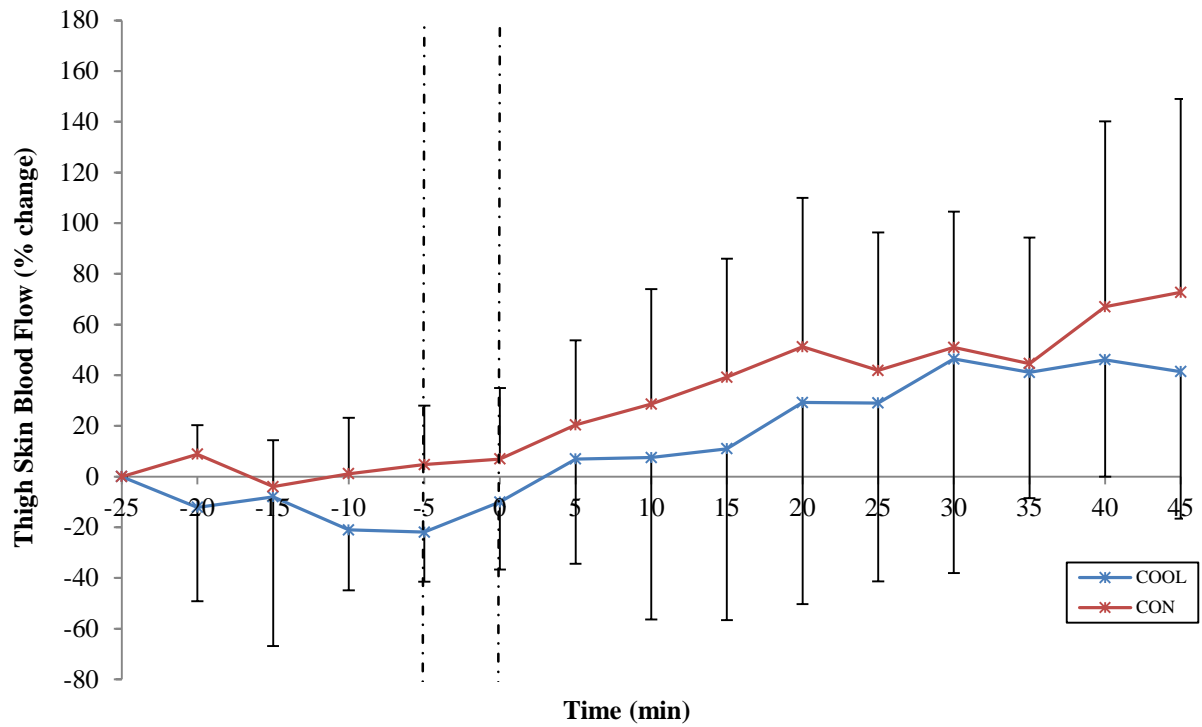


Figure 4.1. Mean \pm SD for thigh SkBF at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min).

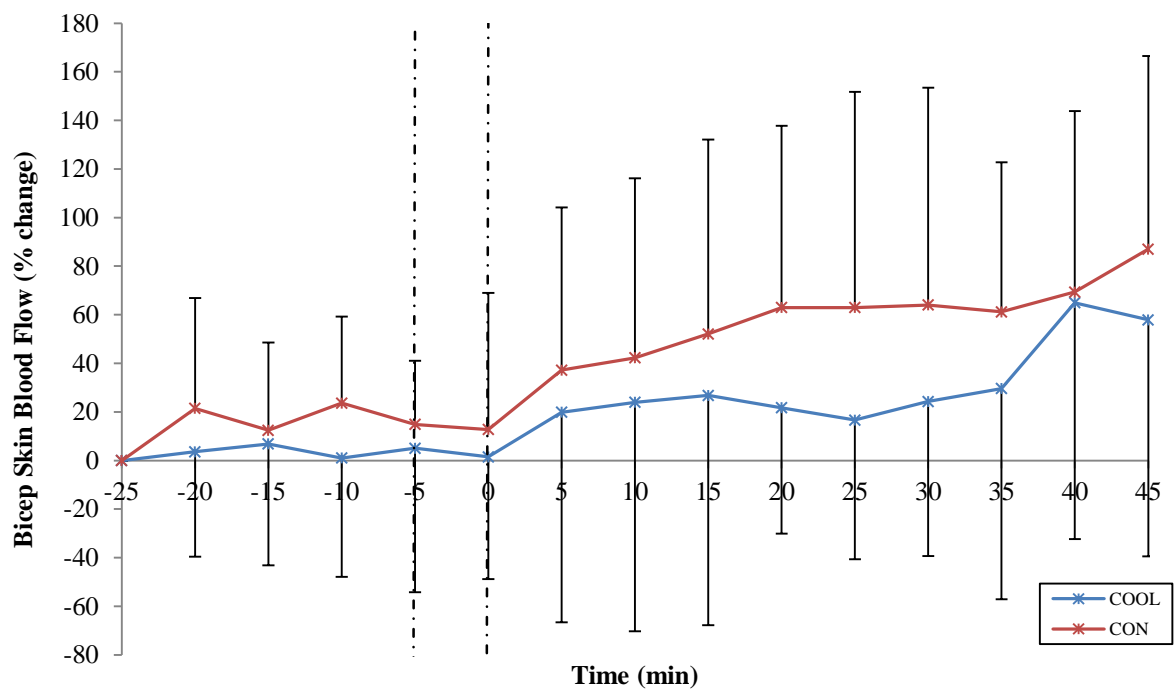


Figure 4.2. Mean \pm SD for bicep SkBF at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min).

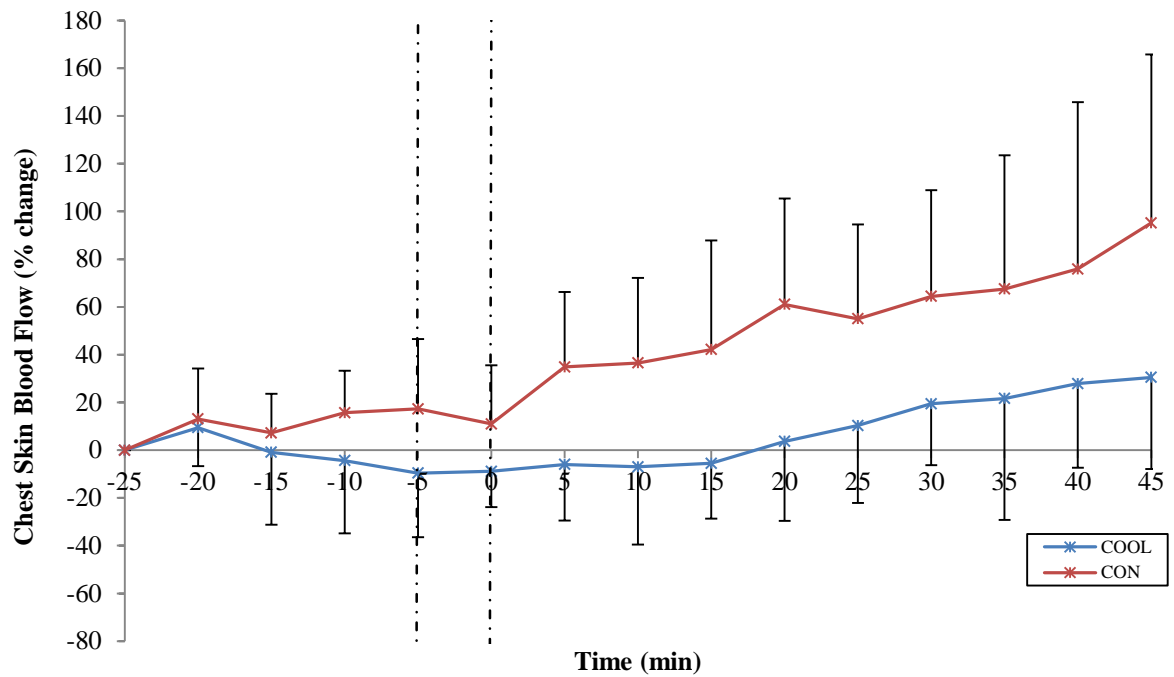


Figure 4.3. Mean \pm SD for chest SkBF at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min).

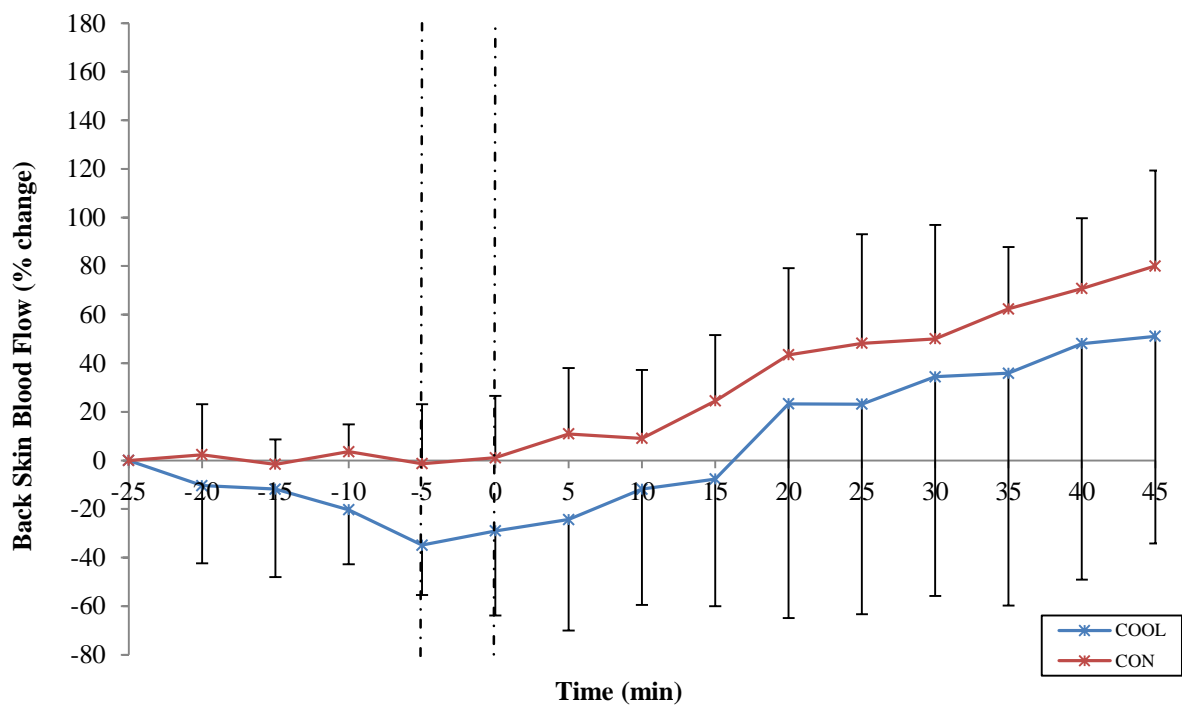


Figure 4.4. Mean \pm SD for back SkBF at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min).

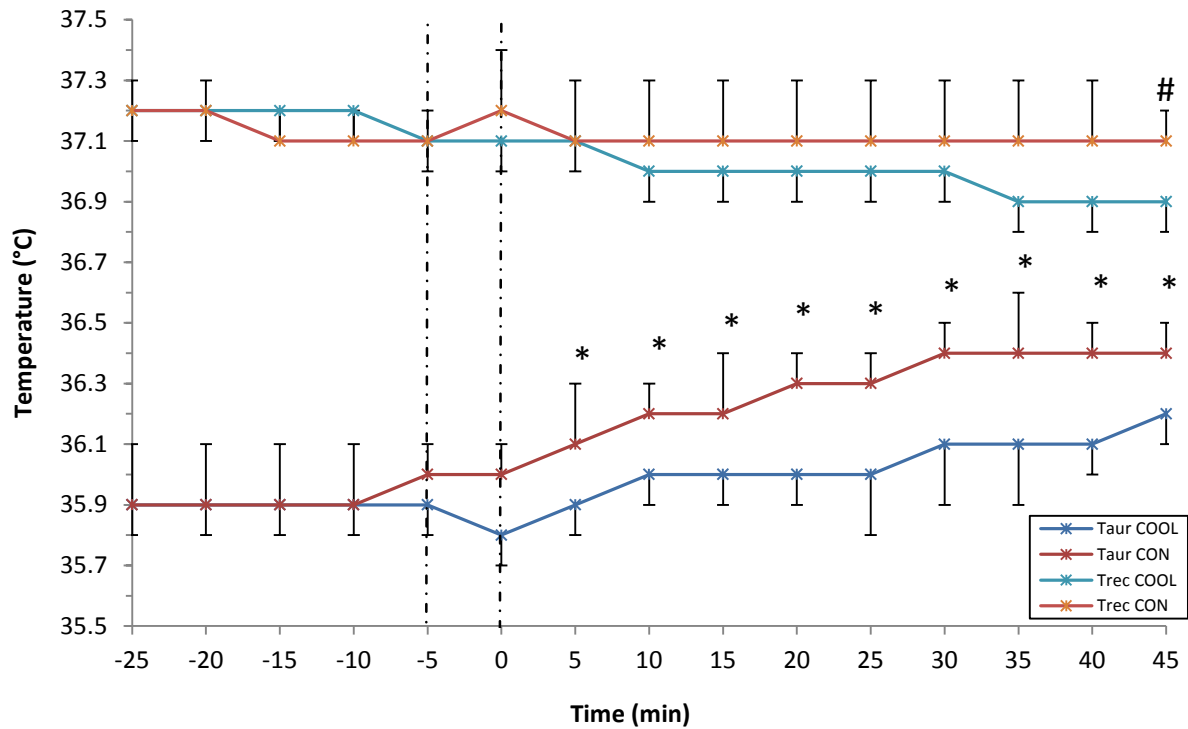


Figure 4.5. Mean \pm SD for T_{aur} and T_{rec} at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min). * = ($P < 0.05$) significant difference between COOL and CON for T_{aur} . # = ($P < 0.05$) significant difference between COOL and CON for T_{rec} .

The T_{rec} responses are shown in Figure 4.5. There was an interaction for trial \times time observed ($P < 0.001$). At baseline values for T_{rec} were similar between both trials ($37.2 \pm 0.1^\circ\text{C}$). At the end of cooling T_{rec} was $37.1 \pm 0.1^\circ\text{C}$ in COOL which was similar to CON. Post-hoc analysis indicated differences between trials on completion of seated rest in the heat (36.9 ± 0.1 and $37.1 \pm 0.2^\circ\text{C}$, for COOL and CON, respectively) ($P < 0.05$).

The T_{aur} responses are shown in Figure 4.5. There was an interaction for trial \times time observed ($P < 0.001$). At baseline values for T_{aur} were similar between trials (35.9 ± 0.1 and $35.9 \pm 0.2^\circ\text{C}$, for COOL and CON, respectively) which remained similar by the end of cooling (35.9 ± 0.1 and $36.0 \pm 0.1^\circ\text{C}$, for COOL and CON, respectively). Post-hoc analysis

indicated differences between trials from 5 to 45 min of seated rest in the heat (36.2 ± 0.1 and $36.4 \pm 0.1^\circ\text{C}$, for COOL and CON at 45 min, respectively) ($P < 0.05$).

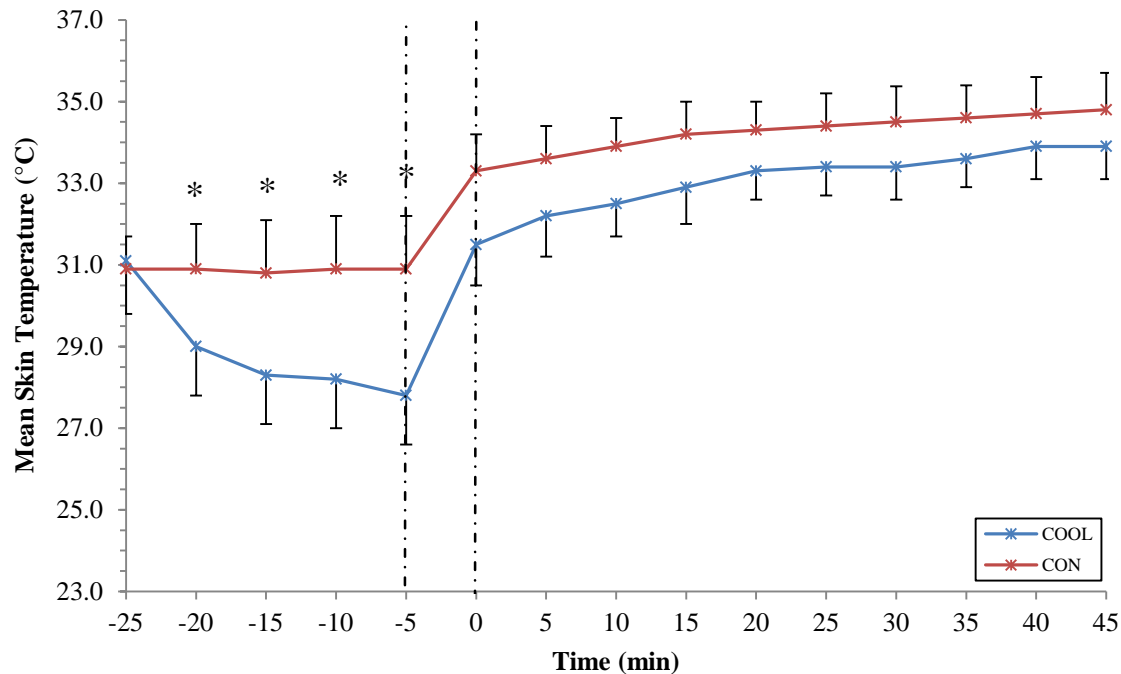


Figure 4.6. Mean \pm SD for mean skin temperature of four sites at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min). * = ($P < 0.05$) significant difference between COOL and CON.

Values for T_{ms} responses are shown in Figure 4.6. There was an interaction for trial \times time observed ($P < 0.001$). Values for T_{ms} were similar at baseline between trials (31.1 ± 1.3 and $30.9 \pm 0.8^\circ\text{C}$, for COOL and CON, respectively). Post-hoc analysis indicated differences between trials at the end of cooling (27.8 ± 1.2 and $30.9 \pm 1.3^\circ\text{C}$, for COOL and CON respectively) ($P < 0.05$). Values for T_{ms} on completion of seated rest in the heat were similar between trials (33.9 ± 0.8 and $34.8 \pm 0.9^\circ\text{C}$, for COOL and CON, respectively).

Values for individual skin temperature responses are shown in Table 4.2. There was an interaction for trial \times time observed for T_{back} ($P<0.001$). Post-hoc analysis indicated differences between trials for T_{back} at the end of cooling ($P<0.05$) which remained significantly cooler at the end of seated rest in the heat (45 min) compared to CON ($P<0.05$).

Table 4.2. Mean \pm SD skin temperature measures ($^{\circ}\text{C}$) at baseline (-25 min), end of cooling (-5 min) and end of seated rest in the heat (45 min). * = ($P<0.05$) significant difference between COOL and CON.

		-25 min	-5 min	45 min
T_{back} ($^{\circ}\text{C}$)	COOL	30.8 \pm 1.0	22.2 \pm 3.6*	33.8 \pm 1.2*
	CON	30.2 \pm 1.3	30.1 \pm 1.3	35.2 \pm 0.9
T_{bicep} ($^{\circ}\text{C}$)	COOL	31.8 \pm 1.2	30.6 \pm 0.6	34.6 \pm 0.7
	CON	31.7 \pm 2.2	31.9 \pm 1.8	35.2 \pm 1.1
T_{thigh} ($^{\circ}\text{C}$)	COOL	31.2 \pm 1.0	30.6 \pm 1.4	29.3 \pm 0.9
	CON	31.5 \pm 1.1	31.8 \pm 1.2	29.7 \pm 1.7
T_{calf} ($^{\circ}\text{C}$)	COOL	30.4 \pm 0.8	29.3 \pm 0.9	33.0 \pm 1.5
	CON	30.1 \pm 1.5	29.7 \pm 1.7	33.9 \pm 0.9
T_{ms} ($^{\circ}\text{C}$)	COOL	31.1 \pm 0.8	27.8 \pm 1.3*	33.9 \pm 0.9
	CON	30.9 \pm 1.3	30.9 \pm 1.2	34.8 \pm 0.8

4.1.2 Perceptual Responses

There was no significant interaction observed for RTE between trials ($P=0.72$) and no significant main effect for time ($P=0.49$). Values at baseline were 4.1 \pm 1.1 and 4.5 \pm 0.7 for COOL and CON, respectively. At the end of cooling (4.1 \pm 0.9 and 4.4 \pm 1.1, for COOL and CON, respectively) and on completion of seated rest in the heat (4.2 \pm 0.8 and 4.3 \pm 0.8, for COOL and CON, respectively) values for RTE were similar.

There were no significant interactions observed in any of the subscales in the POMS questionnaire (anger $P=0.74$, fatigue $P=0.15$, vigour $P=0.44$, depression $P=0.39$, tension $P=0.80$, confusion $P=0.39$). There was no significant main effect for time in any subscale (anger $P=0.74$, fatigue $P=0.25$, vigour $P=0.94$, depression $P=0.39$; tension $P=0.53$; confusion $P=0.39$). Mean values for each subscale are show in Table 4.3.

Table 4.3. Mean \pm SD values for each POMS subscale taken at baseline (-25 min), end of cooling (-5 min) and end of seated rest (45 min).

		-25 min	-5 min	45 min
Anger	COOL	0.1 \pm 0.4	0.1 \pm 0.4	0.0 \pm 0.0
	CON	0.4 \pm 1.1	0.3 \pm 0.7	0.3 \pm 0.5
Fatigue	COOL	0.5 \pm 0.5	0.4 \pm 0.5	0.5 \pm 0.8
	CON	0.3 \pm 0.5	0.8 \pm 1.2	0.8 \pm 0.9
Vigour	COOL	2.3 \pm 1.0	2.4 \pm 0.9	2.5 \pm 0.9
	CON	2.4 \pm 1.1	2.4 \pm 1.2	2.3 \pm 0.9
Depression	COOL	0.0 \pm 0.0	0.0 \pm 0.0	0.0 \pm 0.0
	CON	0.1 \pm 0.4	0.0 \pm 0.0	0.0 \pm 0.0
Tension	COOL	0.4 \pm 0.7	0.1 \pm 0.4	0.3 \pm 0.5
	CON	0.5 \pm 1.1	0.4 \pm 0.7	0.4 \pm 0.7
Confusion	COOL	0.0 \pm 0.0	0.1 \pm 0.4	0.0 \pm 0.0
	CON	0.0 \pm 0.0	0.0 \pm 0.0	0.0 \pm 0.0

Values for RPTS are shown in Figure 4.7. There was an interaction for trial \times time observed ($P<0.001$). Values for RPTS between trials were similar at baseline (3.6 ± 0.4 and 3.4 ± 0.4 , for COOL and CON, respectively). Post-hoc analysis indicated differences between trials at each time point during cooling (2.3 ± 0.6 and 3.6 ± 0.6 , for COOL and CON at the end of cooling, respectively) ($P<0.05$). Values for RPTS on completion of seated rest in the heat were similar between COOL and CON (4.7 ± 0.7 and 5.3 ± 0.8 , respectively).

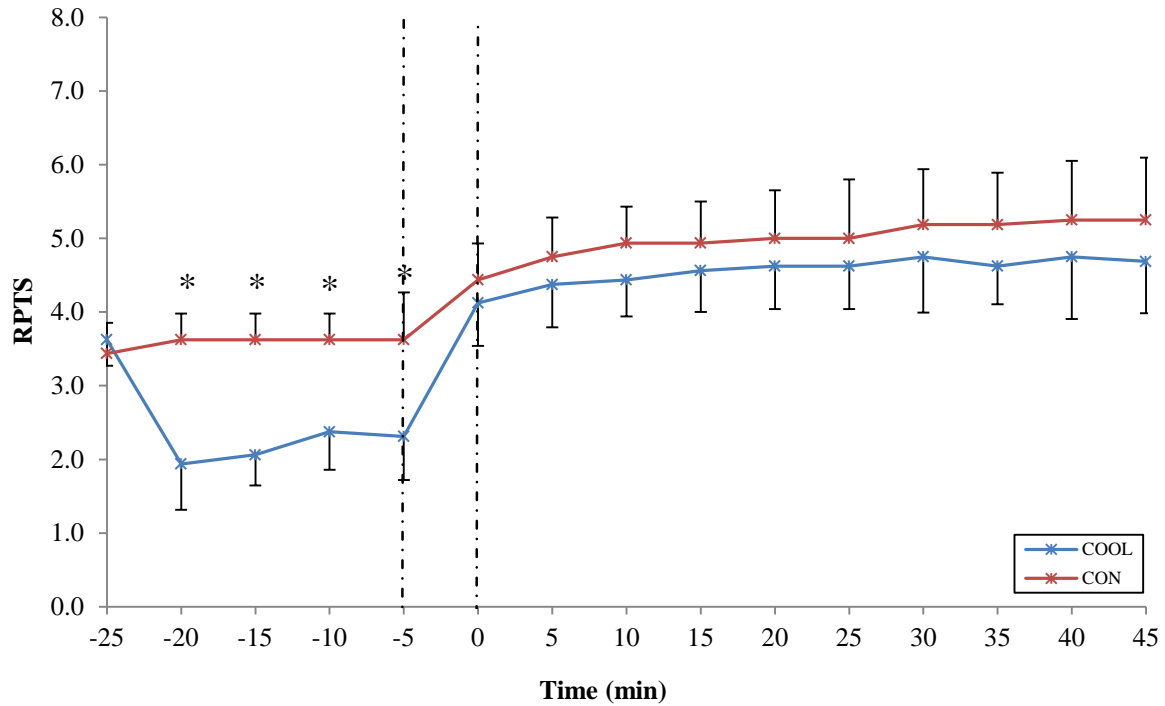


Figure 4.7. Mean \pm SD for RPTS at baseline (-25 min), during cooling (-20 to -5 min) and seated rest in the heat (0 to 45 min). * = ($P < 0.05$) significant difference between COOL and CON.

4.2 Exercise Trials

4.2.1 Physiological Responses

There was no significant interaction observed between trials for $\dot{V}O_2$ ($P = 0.51$) although there was a significant main effect for time ($P < 0.001$). $\dot{V}O_2$ at baseline was 0.41 ± 0.06 and 0.38 ± 0.06 L.min⁻¹ for COOL and CON, respectively. On completion of exercise (40-41 min recorded) values for $\dot{V}O_2$ were 1.75 ± 0.41 and 1.63 ± 0.46 L.min⁻¹ for COOL and CON, respectively, which represented 52.2 and 49.5% of $\dot{V}O_{2max}$, respectively. Values for $\dot{V}O_2$ on completion of the PT were 2.74 ± 0.81 and 2.50 ± 0.80 L.min⁻¹ for COOL and CON, respectively, which represented 82.9 and 74.7% of $\dot{V}O_{2max}$, respectively.

The HR response during exercise is shown in Figure 4.8. There was no significant interaction ($p=0.98$) however, there was a significant main effect for time ($P<0.001$). Values for HR at baseline were similar between trials (71 ± 7 and 68 ± 6 beats.min⁻¹, for COOL and CON, respectively). Values for HR at the end of cooling were similar between COOL and CON (67 ± 6 and 68 ± 5 beats.min⁻¹, respectively). Values increased to 149 ± 10 and 145 ± 14 beats.min⁻¹ at the end of exercise and increased further to 181 ± 10 and 180 ± 8 beats.min⁻¹ for COOL and CON, respectively, at the end of the PT.

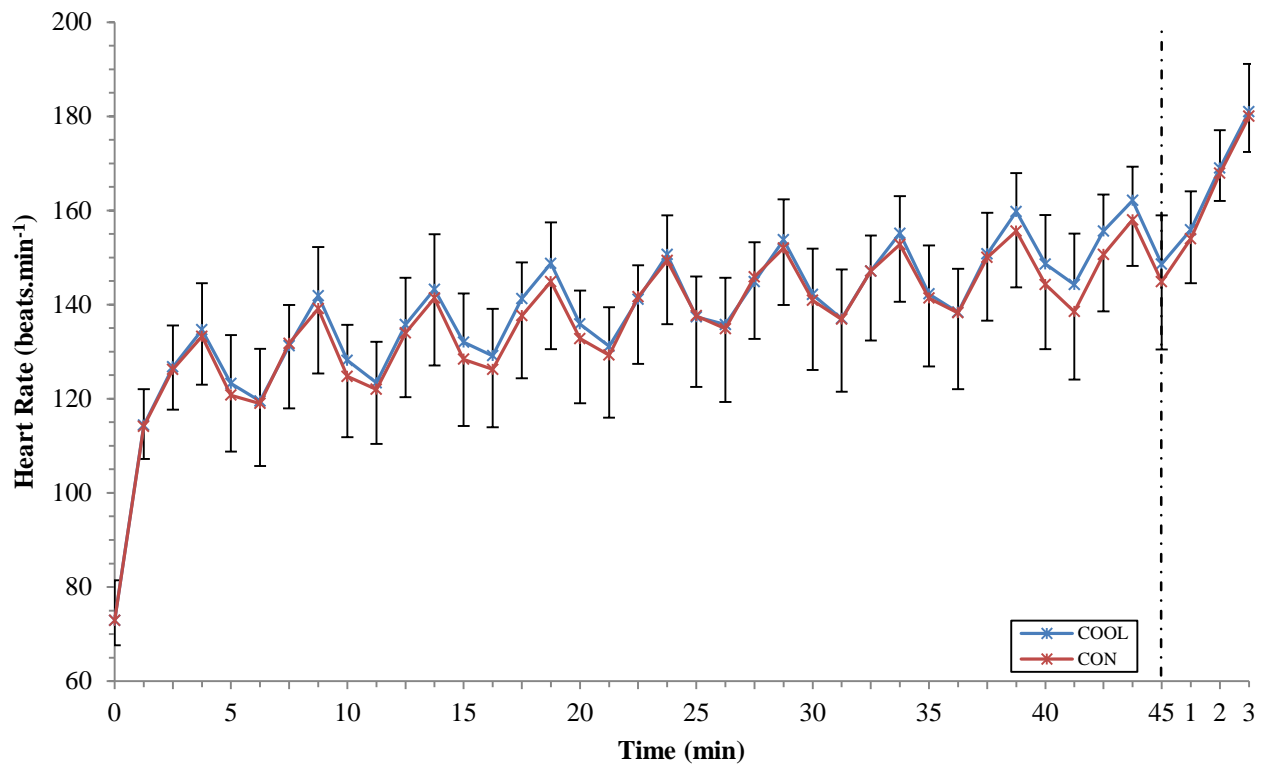


Figure 4.8. Mean \pm SD for HR after 5 min of seated rest in the heat (0 min), during exercise (0 to 45 min) and PT (1, 2 and 3). Numbers 1, 2 and 3 relates to each individual km of the PT.

No significant interaction was observed between trials for [BLa] ($P=0.90$) however, there was a significant main effect for time ($P<0.001$). [BLa] values at baseline were 1.06 ± 0.20 and

0.91 \pm 0.33 mmol.l⁻¹ for COOL and CON, respectively. On completion of exercise [BLa] was 3.31 \pm 1.02 and 3.00 \pm 1.11 mmol.l⁻¹ for COOL and CON, respectively. On completion of the PT [BLa] was 7.35 \pm 2.70 and 6.58 \pm 1.08 mmol.l⁻¹ for COOL and CON, respectively. No significant interactions were observed for [Hb] (P=0.66) or Hct (P=0.76) between trials (Table 4.4). However, there was a significant main effect for time for Hct (P=0.05) but not [Hb] (P=0.07) resulting in non significant changes in plasma volume (1.62 \pm 14.24 and -1.03 \pm 3.92%, for COOL and CON, respectively) (P=0.63). The total amount of body mass lost between COOL (0.7 \pm 0.0 kg) and CON (0.7 \pm 0.1 kg) was not significantly different (P=0.93) although there was a significant main effect for time (P<0.001). Sweat rate (L.h⁻¹) did not significantly differ between COOL (0.98 \pm 0.43 L.h⁻¹) and CON (0.97 \pm 0.51 L.h⁻¹) (P=0.94).

Table 4.4. Mean \pm SD for [BLa], [Hb] and Hct at baseline (-25 min), end of exercise (45 min) and end of the PT (50 min).

		-25 min	45 min	50 min
[BLa] mmol.l ⁻¹	COOL	0.93 \pm 0.33	3.00 \pm 1.08	6.58 \pm 1.08
	CON	1.06 \pm 0.20	3.31 \pm 1.02	7.35 \pm 2.70
[Hb] d.L ⁻¹	COOL	150 \pm 15	-	155 \pm 15
	CON	151 \pm 12	-	159 \pm 14
Hct %	COOL	45 \pm 2	-	46 \pm 3
	CON	45 \pm 3	-	47 \pm 3

SkBF for the thigh, bicep, chest and back during cooling, exercise and PT in COOL and CON are shown in Figures 4.9 to 4.12, respectively. All SkBF sites measured followed the same trend with no significant interaction between trials at any time point. There was a significant main effect for time for the thigh (P<0.001), bicep (P<0.001), chest (P<0.001) and back (P<0.001).

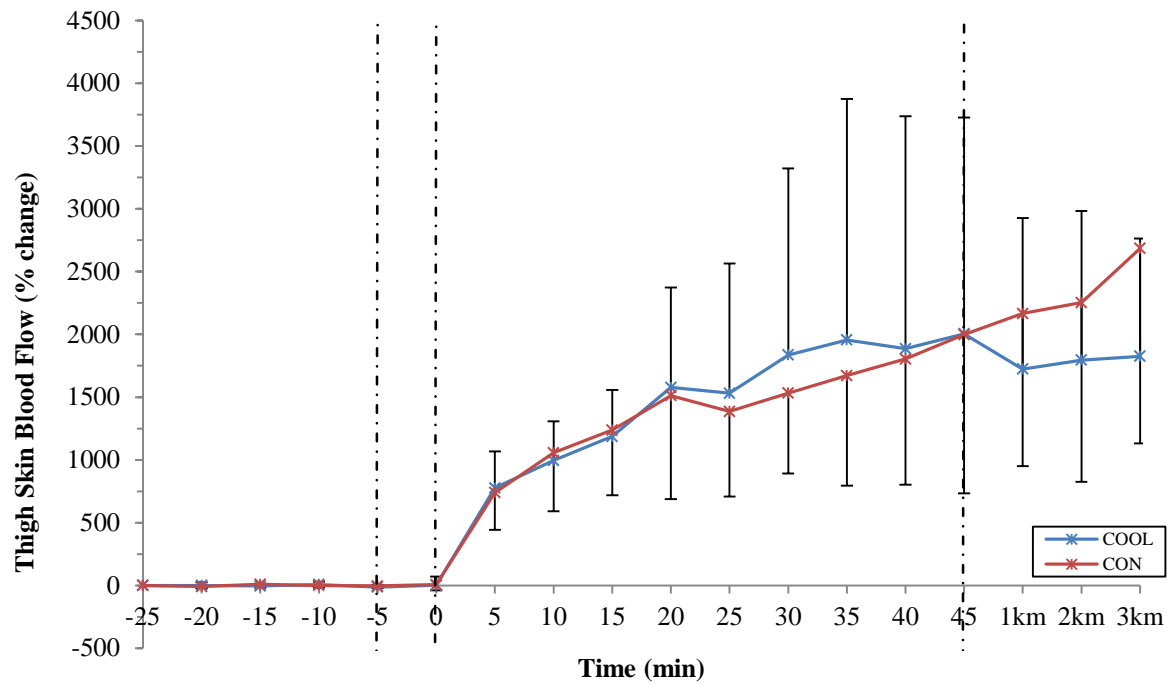


Figure 4.9. Mean \pm SD for thigh SkBF at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

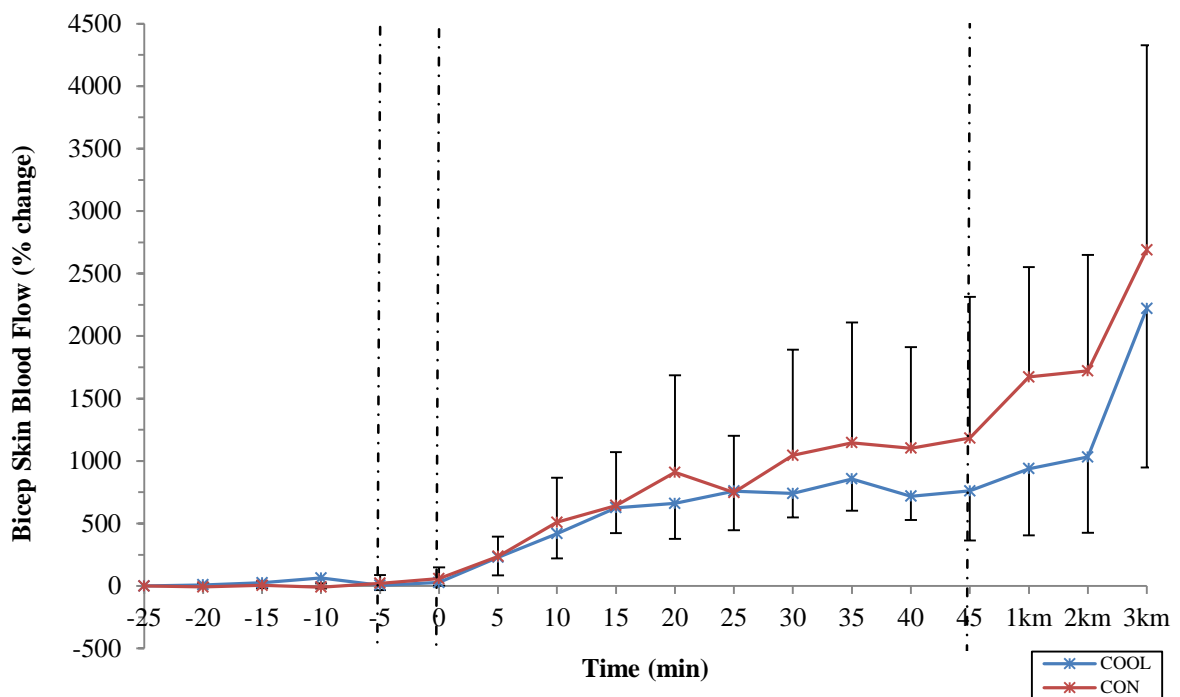


Figure 4.10. Mean \pm SD for bicep SkBF at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

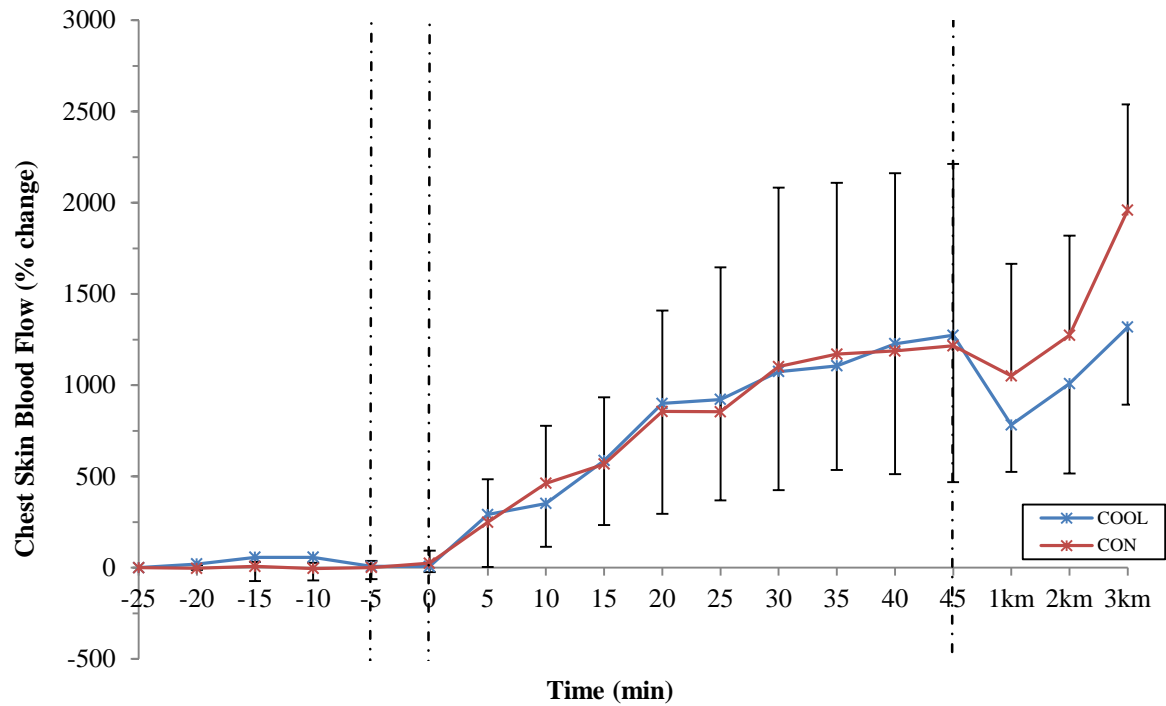


Figure 4.11. Mean \pm SD for chest SkBF at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

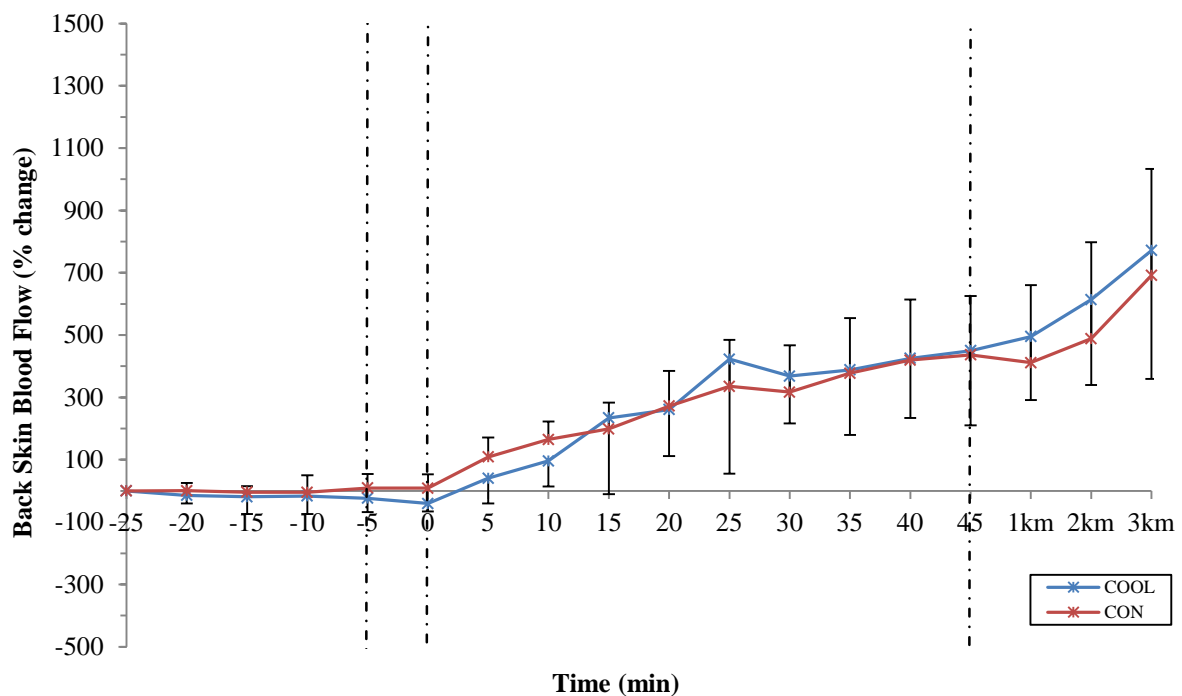


Figure 4.12. Mean \pm SD for back SkBF at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

In order to demonstrate differences between SkBF responses during seated rest and exercise trials, Figure 4.13 demonstrates SkBF for the back in all four experimental trials. There was an interaction for trial \times time observed between exercise in COOL (EX COOL) and seated rest in the heat in COOL (SR COOL) ($P < 0.001$) and between exercise in CON (EX CON) and seated rest in the heat in CON (SR CON) ($P < 0.001$). Post-hoc analysis indicated differences between the seated rest and exercise trials which differed from 5-45 min in CON ($P < 0.05$). In addition, the seated rest and exercise trials differed from 15-45 min in COOL ($P < 0.05$).

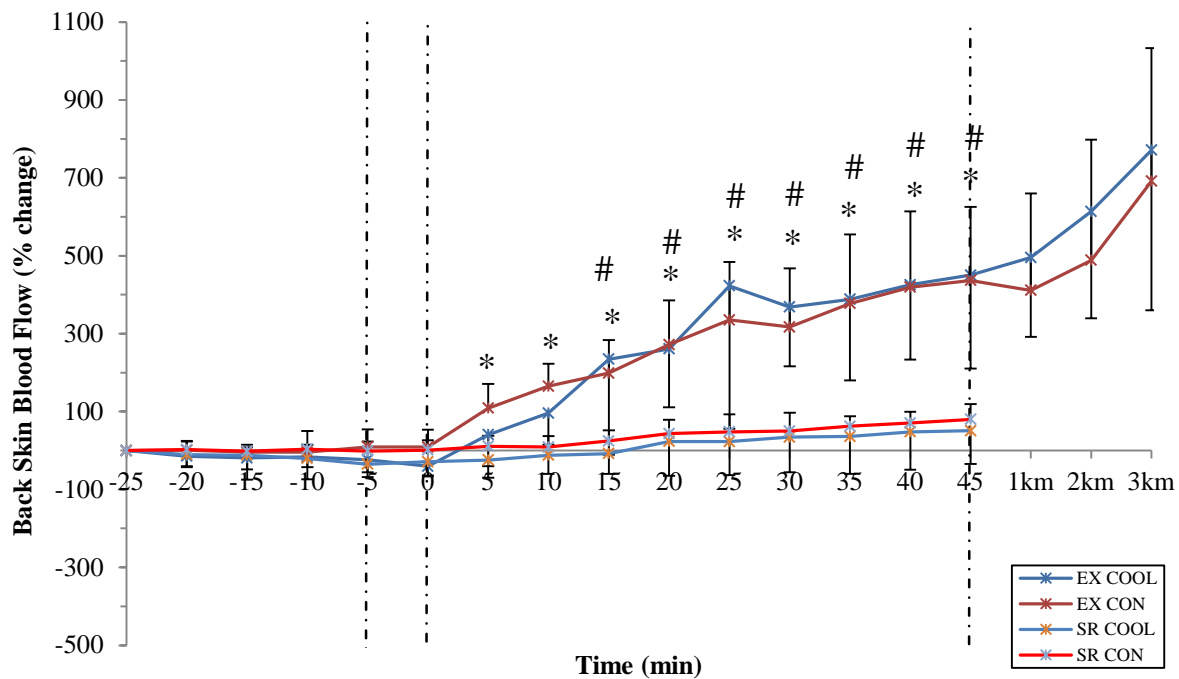


Figure 4.13. Mean \pm SD for back SkBF during exercise and seated rest protocols in both COOL and CON. * = ($P < 0.05$) significant difference between exercise and seated rest in the heat in CON. # = ($P < 0.05$) significant difference between exercise and seated rest in the heat in COOL.

To demonstrate regional differences in SkBF all four SkBF sites during exercise in CON only were plotted and are shown in Figure 4.14. There was an interaction for trial \times time observed ($P<0.001$). Post-hoc analysis indicated differences between thigh and back SkBF at 45 min of exercise and throughout the PT ($P<0.05$). No other SkBF sites significantly differed from one another at any time point.

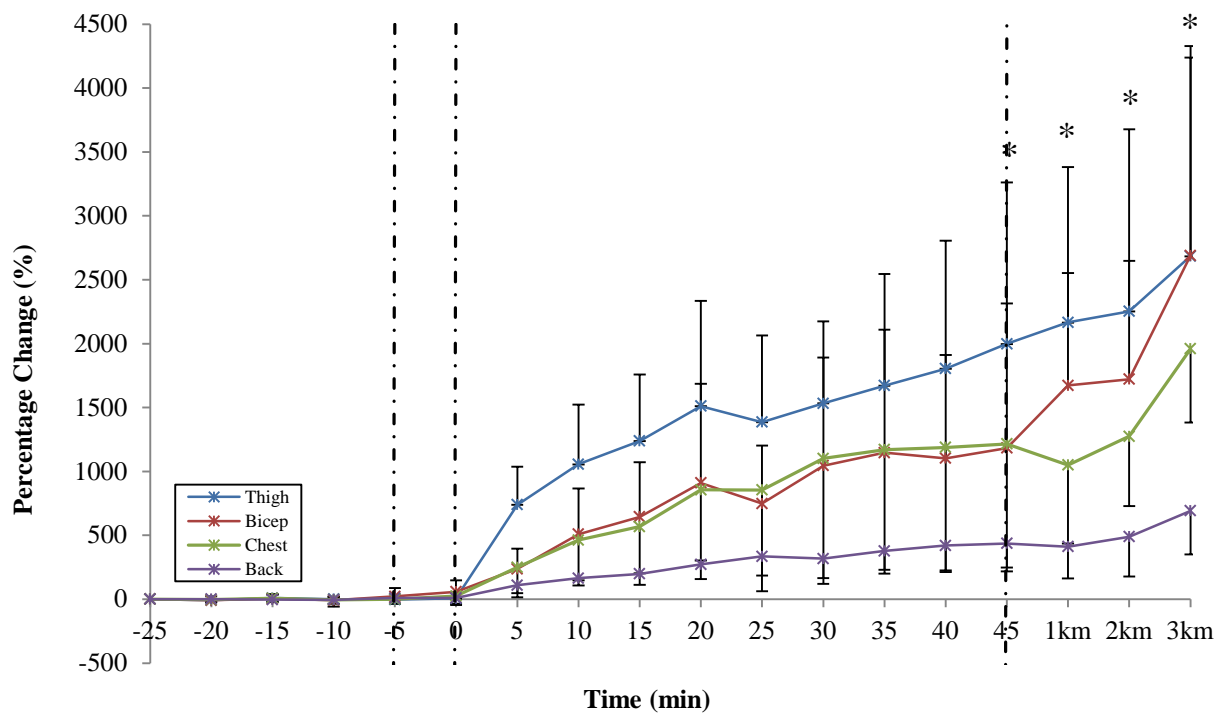


Figure 4.14. Mean \pm SD SkBF at all four measured sites during cooling (-20 to -5 min), exercise(0 to 45 min) and PT in CON only. * = ($P<0.05$) significant difference between thigh and back SkBF.

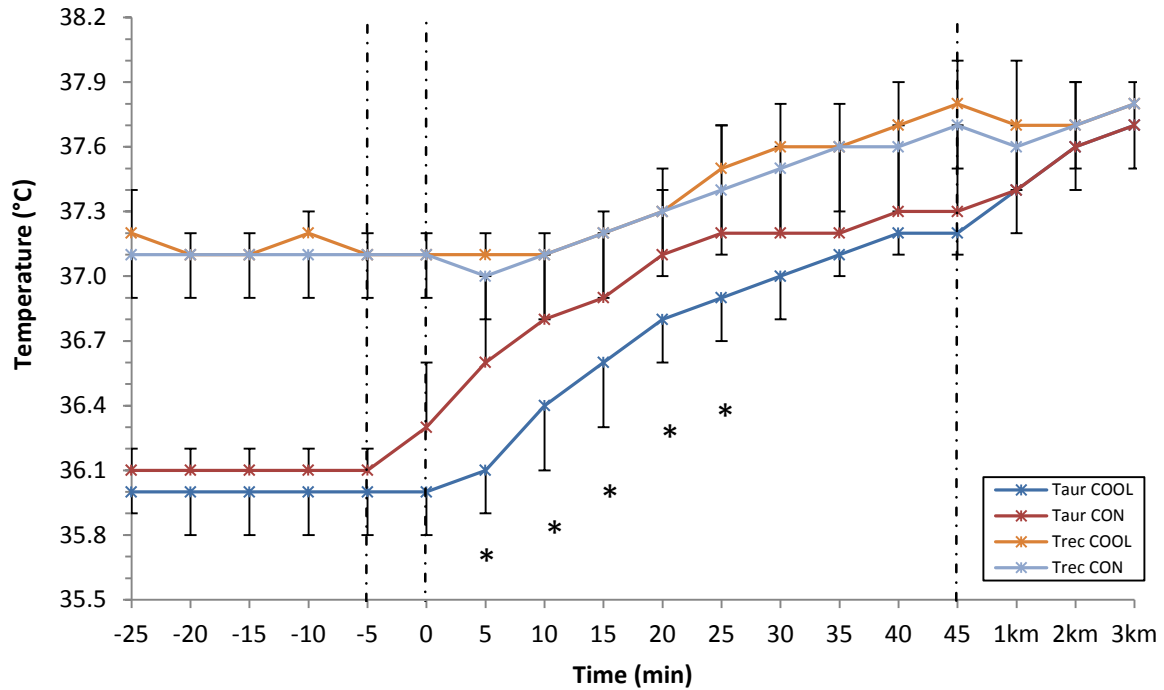


Figure 4.15. Mean \pm SD for T_{aur} and T_{rec} ($^{\circ}$ C) at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT. * = ($P < 0.05$) significant difference between T_{aur} COOL and T_{aur} CON.

Values for T_{rec} are shown in Figure 4.15. No significant interaction was observed between trials ($P = 0.99$) however, there was a significant main effect for time ($P < 0.001$). Values for T_{rec} at baseline were similar between trials (37.1 ± 0.2 and $37.2 \pm 0.2^{\circ}$ C, for COOL and CON, respectively) and after cooling (37.1 ± 0.1 and $37.1 \pm 0.2^{\circ}$ C for COOL and CON, respectively). At the end of exercise values for T_{rec} were 37.8 ± 0.3 and $37.7 \pm 0.2^{\circ}$ C for COOL and CON, respectively and remained at these levels at the end of the PT (both $37.8 \pm 0.2^{\circ}$ C).

Values for T_{aur} are shown in Figure 4.15. There was an interaction for trial \times time observed ($P < 0.001$). Values for T_{aur} were similar between trials at baseline (36.1 ± 0.1 and $36.0 \pm 0.1^{\circ}$ C, for COOL and CON, respectively) and after cooling (36.1 ± 0.1 and $36.0 \pm 0.2^{\circ}$ C, for

COOL and CON, respectively). Post-hoc analysis indicated differences between trials from 5-25 min ($P<0.05$). On completion of exercise values for T_{aur} were similar between COOL and CON (37.3 ± 0.4 and $37.2 \pm 0.1^{\circ}\text{C}$, respectively). Values for T_{aur} on completion of the PT were identical between trials ($37.7 \pm 0.2^{\circ}\text{C}$).

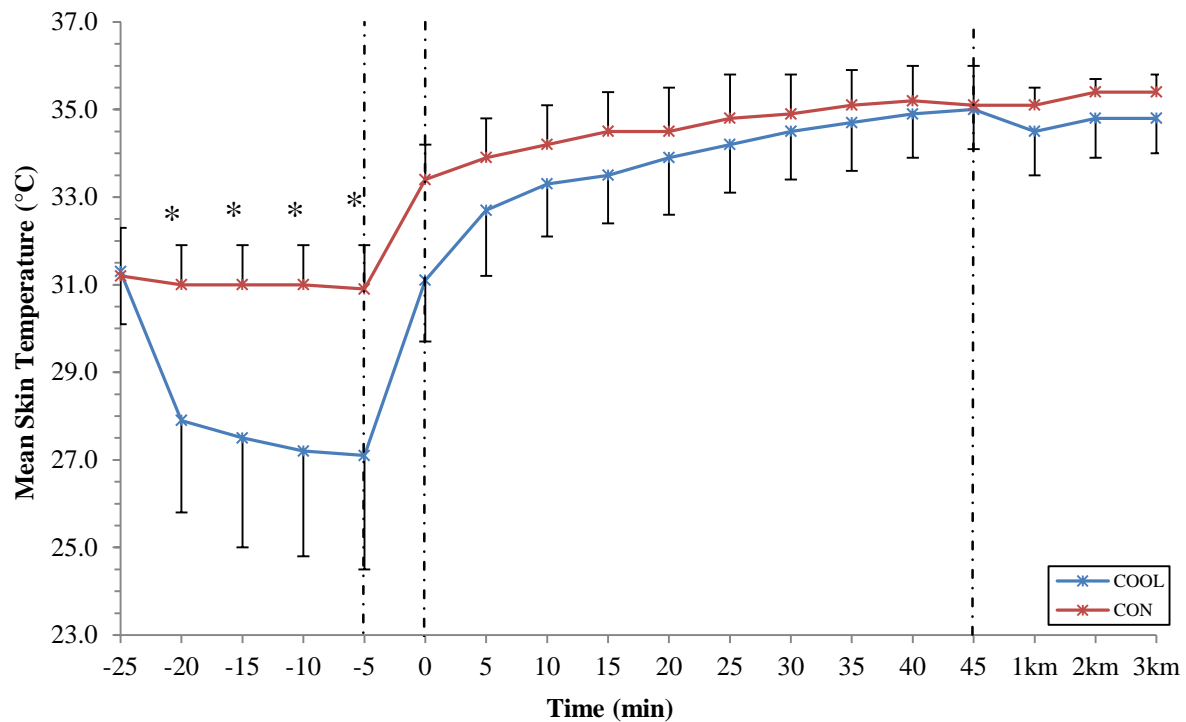


Figure 4.16. Mean \pm SD for mean skin temperature ($^{\circ}\text{C}$) for four sites measured at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

The T_{ms} responses are shown in Figure 4.16. There was an interaction for trial \times time observed ($P<0.001$). Values for T_{ms} at baseline were similar between trials (31.3 ± 1.2 and $31.2 \pm 1.1^{\circ}\text{C}$, for COOL and CON, respectively). Post-hoc analysis indicated differences between trials at each time point during cooling (27.1 ± 2.6 and $30.9 \pm 1.0^{\circ}\text{C}$, at the end of cooling, for COOL and CON, respectively) ($P<0.05$). Values for T_{ms} were similar between COOL and CON on completion of exercise (35.0 ± 0.9 and $35.1 \pm 0.9^{\circ}\text{C}$, respectively) and the PT (34.8 ± 0.8 and $35.4 \pm 0.4^{\circ}\text{C}$, respectively).

T_{back} and T_{bicep} responses are shown in Figure 4.17 and 4.18, respectively. There was an interaction for trial \times time observed for both T_{back} ($P<0.001$) and T_{bicep} ($P=0.04$). Both T_{back} and T_{bicep} values at baseline were similar between trials but after cooling post-hoc analysis indicated differences between trials ($P<0.05$). T_{back} at the end of cooling was 20.3 ± 5.0 and $30.4 \pm 1.6^{\circ}\text{C}$ for COOL and CON, respectively ($P<0.05$). T_{bicep} at the end of cooling was 29.3 ± 3.9 and $31.9 \pm 0.8^{\circ}\text{C}$ for COOL and CON, respectively ($P<0.05$). T_{back} remained significantly lower in COOL compared to CON up to 15 min of exercise whereas T_{bicep} did not significantly differ between trials during exercise. T_{back} and T_{bicep} values were similar between trials at the end of exercise and the PT ($P>0.05$). Table 4.5 shows individual skin temperature sites.

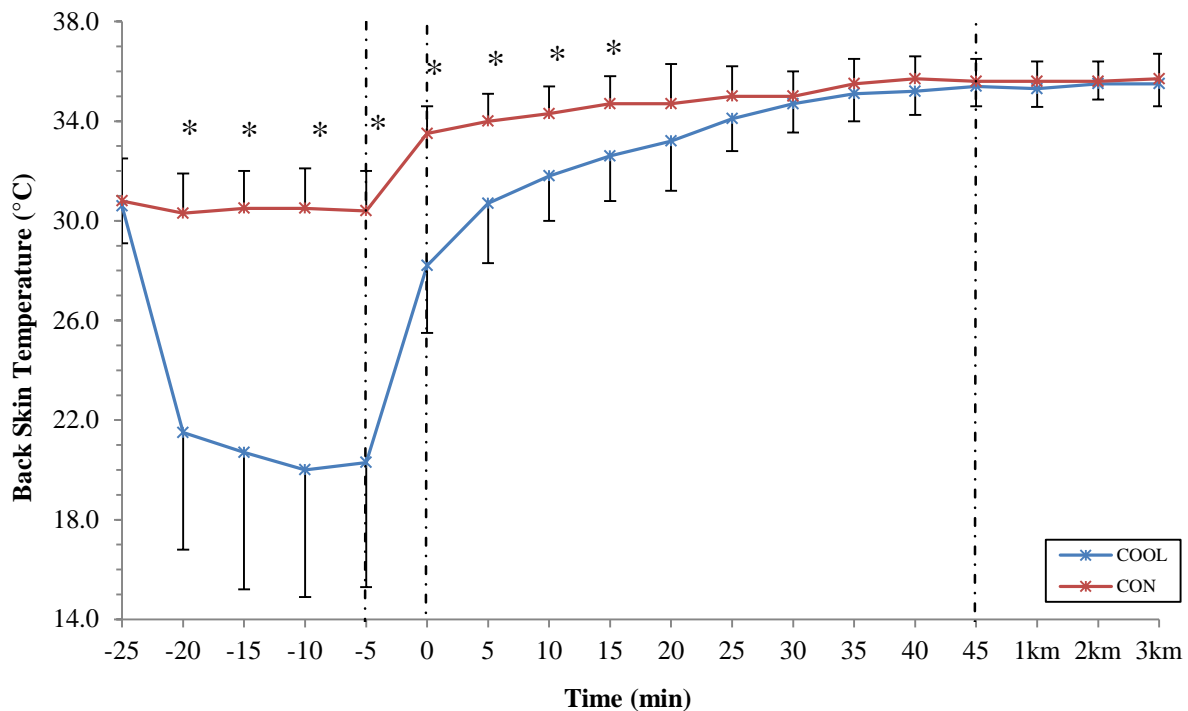


Figure 4.17. Mean \pm SD for T_{back} ($^{\circ}\text{C}$) at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

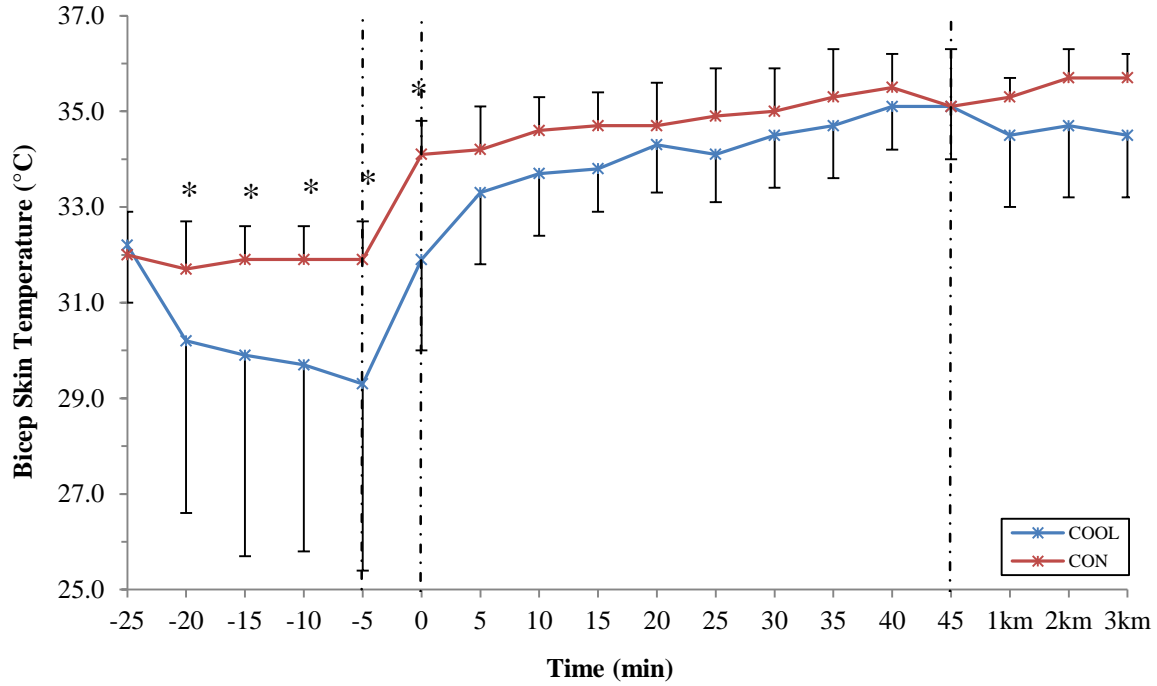


Figure 4.18. Mean \pm SD for T_{bicep} ($^{\circ}\text{C}$) at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT.

Table 4.5. Mean \pm SD skin temperature measures ($^{\circ}\text{C}$) taken at baseline (-25 min), end of cooling (-5 min), end of exercise (45 min) and end of the PT (50 min). * = ($P < 0.05$) significant difference between COOL and CON.

		-25 min	-5 min	45 min	50 min
T_{back} ($^{\circ}\text{C}$)	COOL	30.6 \pm 1.5	20.3 \pm 5.0*	35.4 \pm 0.8	35.5 \pm 0.9
	CON	30.8 \pm 1.7	30.4 \pm 1.6	35.6 \pm 0.9	35.7 \pm 1.0
T_{bicep} ($^{\circ}\text{C}$)	COOL	32.2 \pm 1.2	29.3 \pm 3.9*	35.0 \pm 1.1	34.5 \pm 1.3
	CON	32.0 \pm 0.9	31.9 \pm 0.8	35.1 \pm 1.2	35.7 \pm 0.5
T_{thigh} ($^{\circ}\text{C}$)	COOL	31.7 \pm 1.3	31.4 \pm 1.3	35.3 \pm 1.1	34.8 \pm 1.3
	CON	31.9 \pm 1.2	32.0 \pm 1.1	35.5 \pm 1.1	35.5 \pm 1.2
T_{calf} ($^{\circ}\text{C}$)	COOL	30.4 \pm 2.1	29.6 \pm 2.4	34.3 \pm 1.6	34.3 \pm 1.2
	CON	29.9 \pm 1.3	29.3 \pm 1.2	34.0 \pm 1.2	34.7 \pm 0.6
T_{ms} ($^{\circ}\text{C}$)	COOL	31.3 \pm 1.2	27.1 \pm 2.6*	35.0 \pm 0.9	34.8 \pm 0.8
	CON	31.2 \pm 1.1	30.9 \pm 1.0	35.1 \pm 0.9	35.4 \pm 0.4

4.2.2 Perceptual Responses

There was no significant interaction ($P=0.96$) between trials or main effect for time ($P=0.44$) for RTE. At baseline RTE was 4.4 ± 0.7 and 4.3 ± 0.7 for COOL and CON, respectively. At the end of cooling values were identical to that of baseline (4.4 ± 0.7 and 4.3 ± 0.7 , for COOL and CON, respectively) and prior to the PT values were similar (4.3 ± 0.8 and 4.3 ± 0.7 , for COOL and CON, respectively).

No significant interaction was observed for RPE between trials ($P=0.50$) although there was a significant main effect for time ($P<0.001$). Values for RPE on completion of exercise were 15 ± 2 in both COOL and CON, respectively, and were 19 ± 1 and 18 ± 2 at the end of the PT, respectively. There were no significant interactions observed in any of the subscales in the POMS questionnaire (anger $P=0.26$, fatigue $P=0.08$, vigour $P=0.42$, depression $P=0.39$, tension $P=0.39$, confusion $P=0.13$). There was a tendency ($P=0.08$) for the fatigue subscale to have lower reported values in COOL compared to CON at completion of 45 min of exercise. Only one significant main effect for time was observed for the fatigue subscale ($P<0.001$). Individual values for each subscale are show in Table 4.6.

Table 4.6. Mean \pm SD values for each POMS subscale taken at baseline (-25 min), end of cooling (-5 min) and at the end of exercise (45 min).

		-25 min	-5 min	45 min
Anger	COOL	0.3 \pm 0.7	0.1 \pm 0.4	0.0 \pm 0.0
	CON	0.0 \pm 0.0	0.1 \pm 0.4	0.1 \pm 0.4
Fatigue	COOL	0.6 \pm 0.7	0.4 \pm 0.5	1.9 \pm 1.0
	CON	0.4 \pm 0.5	0.4 \pm 0.5	2.1 \pm 1.1
Vigour	COOL	2.4 \pm 1.3	2.4 \pm 0.7	1.8 \pm 1.0
	CON	2.4 \pm 0.9	2.5 \pm 0.8	2.3 \pm 1.0
Depression	COOL	0.1 \pm 0.4	0.0 \pm 0.0	0.0 \pm 0.0
	CON	0.1 \pm 0.4	0.0 \pm 0.0	0.0 \pm 0.0
Tension	COOL	0.1 \pm 0.4	0.3 \pm 0.5	0.3 \pm 0.7
	CON	0.1 \pm 0.4	0.1 \pm 0.4	0.3 \pm 0.7
Confusion	COOL	0.0 \pm 0.0	0.3 \pm 0.5	0.0 \pm 0.0
	CON	0.0 \pm 0.0	0.0 \pm 0.0	0.0 \pm 0.0

Values for RPTS are shown in Figure 4.19. There was an interaction for trial \times time observed ($P < 0.001$). Values for RPTS at baseline were identical between COOL and CON (3.7 ± 0.4). Post-hoc analysis indicated differences between trials from -20 to -10 min during cooling ($P < 0.05$) but did not significantly differ between trials at the end of cooling (2.6 ± 0.7 and 3.6 ± 0.5 , for COOL and CON, respectively). Values for RPTS between trials on completion of exercise (6.7 ± 0.9 and 6.8 ± 0.9 for COOL and CON, respectively) were similar to values on completion of the PT (6.9 ± 0.9 and 6.9 ± 1.0 , for COOL and CON, respectively).

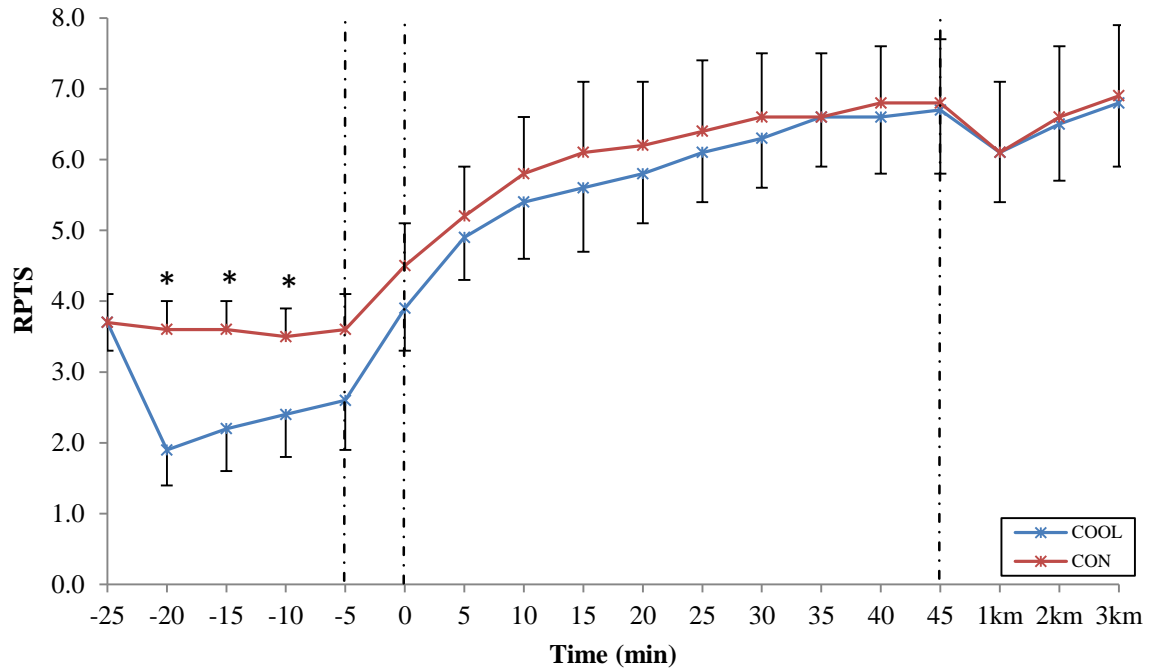


Figure 4.19. Mean \pm SD for RPTS at baseline (-25 min), during cooling (-20 to -5 min), exercise (0 to 45 min) and PT. * = ($P < 0.05$) significant difference between COOL and CON.

4.3 Performance Trial

Completion time of the PT did not significantly differ ($P = 0.81$) between COOL and CON (285 ± 53 and 291 ± 51 seconds, respectively). No significant interaction was observed between trials for cadence ($\text{rev} \cdot \text{min}^{-1}$) ($P = 0.33$) however, there was a significant main effect for time ($P < 0.001$) between 2.5 to 3.0 km. Cadence recorded at completion was 128 ± 18 and 123 ± 11 ($\text{rev} \cdot \text{min}^{-1}$) for COOL and CON, respectively (Figure 4.20).

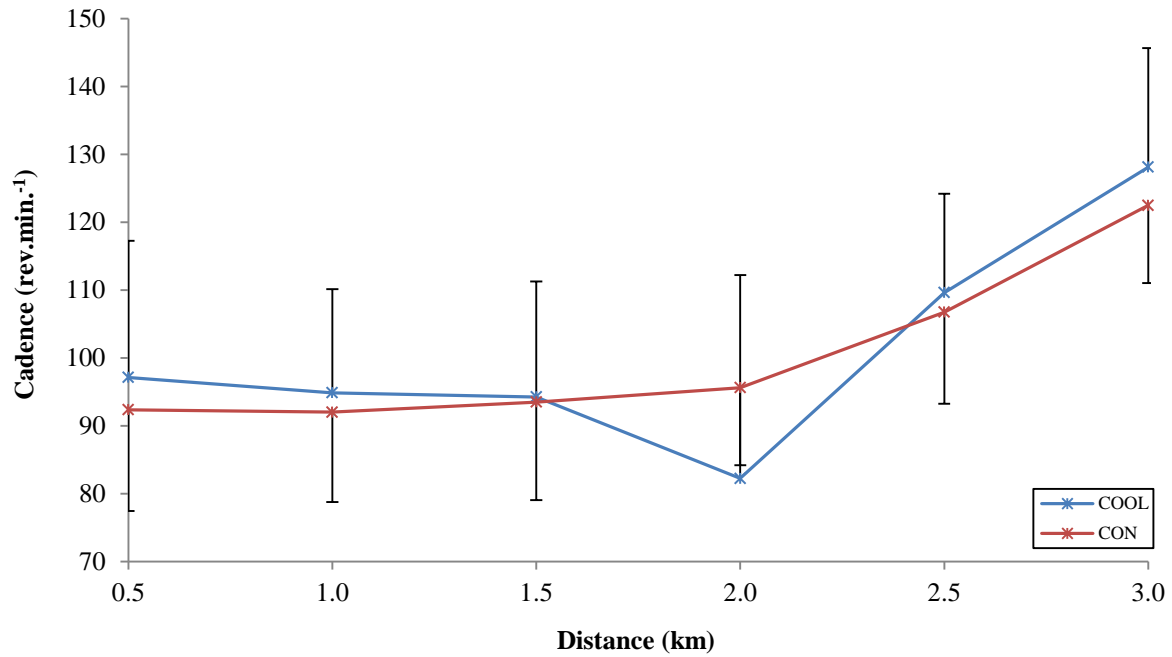


Figure 4.20. Mean \pm SD for cadence (rev.min⁻¹) at each half-kilometre during the PT.

4.4 Reliability of Ice Vest Pre-Cooling Procedure

Values for T_{ms} at rest and during cooling for COOL and CON are shown in Figure 4.21.

Values between seated rest and exercise trials in COOL were consistent and yielded an r value of 0.95. In addition, there was no significant difference between the seated rest and exercise trials in COOL ($P=0.11$). Values for T_{back} at rest and during cooling for COOL and CON are shown in Figure 4.22. Values between seated rest and exercise trials in COOL were consistent and yielded an r value of 0.94. In addition, there was no significant difference between the seated rest and exercise trials in COOL ($P=0.08$).

Values for RPTS at rest and during cooling for COOL and CON are shown in Figure 4.23.

Values between seated rest and exercise trials in COOL were consistent and yielded an r value of 0.98. In addition, there was no significant difference between the seated rest and exercise trials in COOL ($P=0.78$).

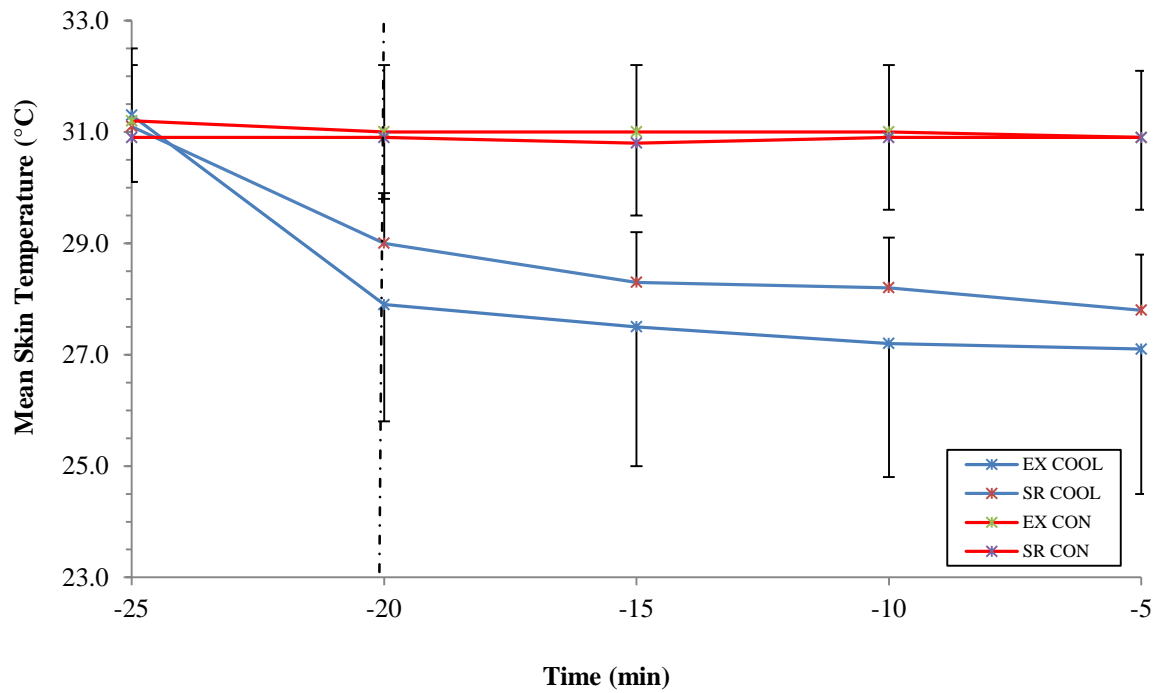


Figure 4.21. Mean \pm SD for mean skin temperature (°C) at baseline (-25 min) and during cooling (-20 to -5 min) for seated rest and exercise trials in both COOL and CON.

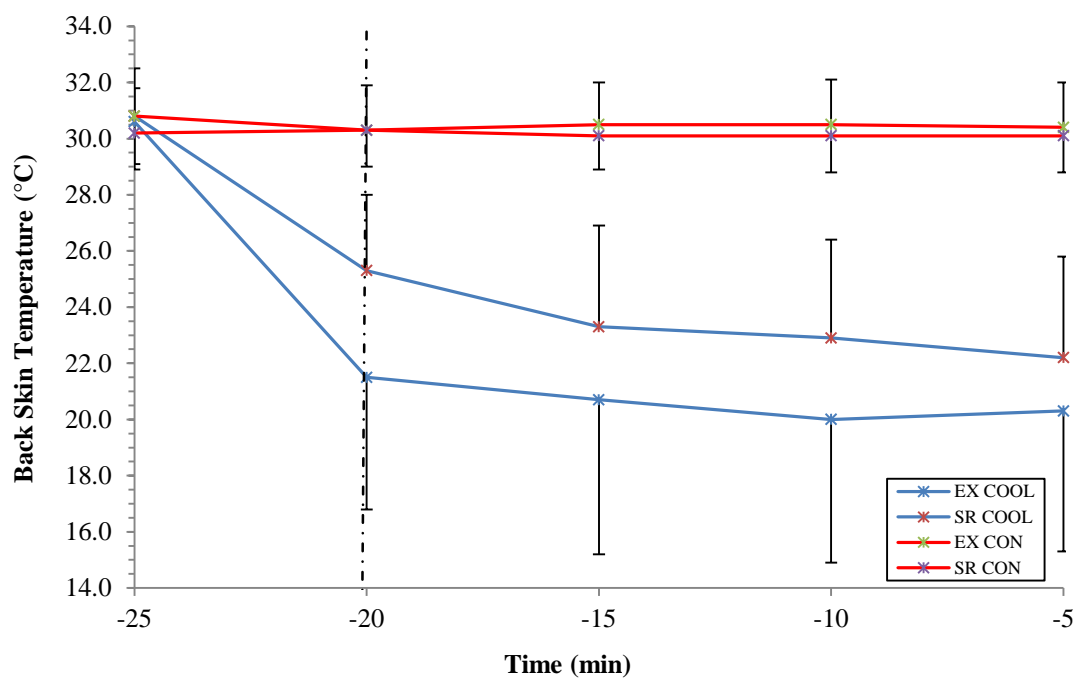


Figure 4.22. Mean \pm SD for back skin temperature (°C) at baseline (-25 min) and during cooling (-20 to -5 min) for seated rest and exercise trials in both COOL and CON.

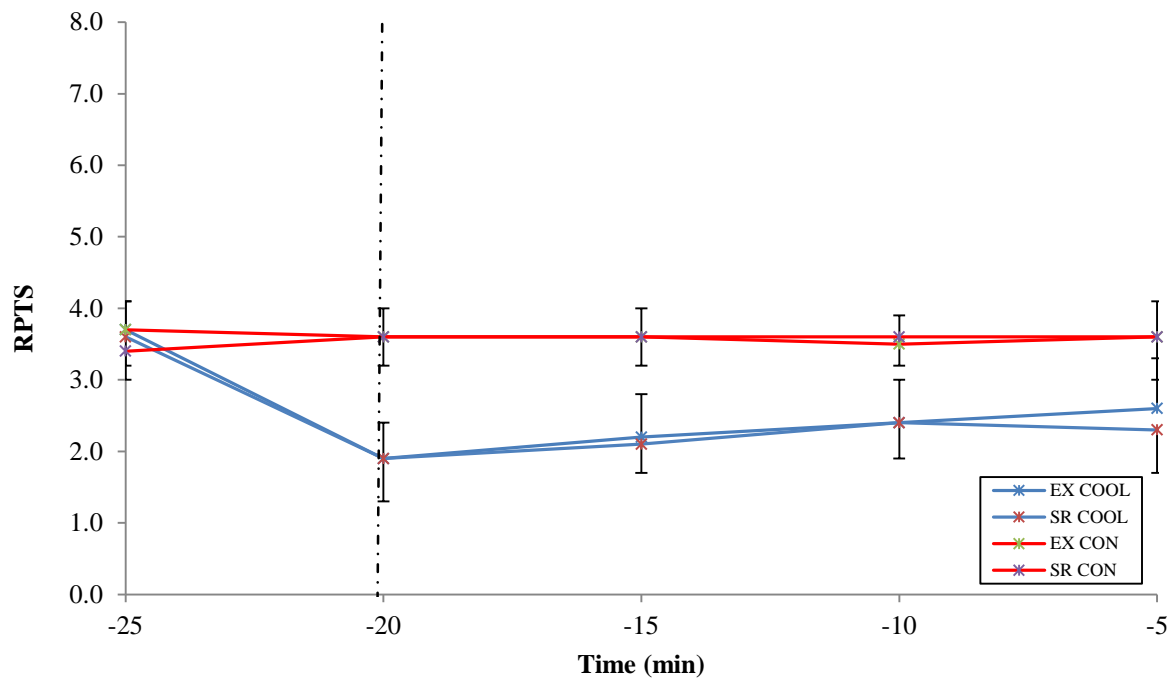


Figure 4.23. Mean \pm SD for RPTS at baseline (-25 min) and during cooling (-20 to -5 min) for seated rest and exercise trials in both COOL and CON.

5.0 Discussion

The main of this study was to determine whether ice vest pre-cooling reduces skin blood flow compared to a no cooling control and subsequently improve exercise performance in the heat. The secondary aim of this study was to determine the effects of pre-cooling on seated rest and heat exposure to observe the responses of cooling without exercise metabolic heat production. The results of this study show that SkBF was not significantly affected during ice vest cooling and during subsequent exercise or a seated rest trial in the heat. There was, however, a significant difference between trials for T_{rec} and T_{aur} during the seated rest trial and a significant difference for T_{aur} only during exercise. Values for T_{ms} and RPTS were significantly lower during cooling but did not significantly differ between trials during the seated rest or exercise trials. Despite changes in thermoregulatory variables ice vest cooling had no significant effect on performance.

5.1 Heart Rate

Mean HR over the exercise period was similar between conditions (138 and 136 beats.min⁻¹, for COOL and CON, respectively) but ranged progressively from 114-162 beats.min⁻¹ across the different exercise intensity bouts. The reason for the observed cardiovascular drift during exercise in the heat is due to combination of increased cutaneous circulation leading to a lower stroke volume (Rowell 1969). In addition, dehydration may also result in a reduction in stroke volume and increased HR (Gonzalez-Alonso et al. 1997) and/or an increased core temperature which directly influences HR (Coyle and Gonzalez-Alonso 2001, Gonzalez-Alonso et al. 1997). Previous research has observed similar HR values following 45 min of intermittent exercise in the heat (Price, Boyd, and Goosey-Tolfrey 2009). However, other research has noted greater values (~157 beats.min⁻¹) during intermittent exercise in the heat

(Duffield et al. 2003). The protocol utilised in the present was similar, but not identical, to Price, Moss, and Rance (2003) which observed average HR values of $148 \text{ beats} \cdot \text{min}^{-1}$ across 30 min of exercise in normothermic conditions. The present study produced marginally lower values for HR compared to other intermittent protocols. In addition, T_{rec} in the present study had only risen 0.5°C above baseline at 35 min of exercise which in contrast to that of Price, Boyd and Goosey-Tolfrey (2009) which observed T_{rec} to be 1.2°C greater than baseline. This shows that the exercise protocol utilised in the present study provided lower levels of physiological and thermoregulatory strain compared to previous research.

HR following the PT was $181 \pm 9 \text{ beats} \cdot \text{min}^{-1}$ which was similar to HR_{max} values found in the preliminary trial ($184 \pm 7 \text{ beats} \cdot \text{min}^{-1}$). These results show that participants were at or very close to maximal exertion on completion of the PT. HR during the seated rest trial did not significantly differ between trials at any time point and remained relatively stable throughout ($70 \pm 2 \text{ beats} \cdot \text{min}^{-1}$).

5.2 Blood Lactate

During exercise values for [BLa] were sub-maximal and represented aerobic metabolism. Krstrup et al. (2006) reported that blood lactate levels during football matches were between $5\text{-}6 \text{ mmol} \cdot \text{l}^{-1}$ which is greater than the values observed during exercise in the present study which were maintained at $4 \text{ mmol} \cdot \text{l}^{-1}$ throughout exercise. In addition, Price, Moss, and Rance (2003) observed that [BLa] values increased to $\sim 11 \text{ mmol} \cdot \text{l}^{-1}$. Even though the present study's exercise protocol was adapted from the latter study, no short duration maximal sprints were included. The rationale for this was because the aim of the present study exercise protocol was to increase body temperature over 45 min and not to exhaust participants. Future research could utilise an exercise protocol that would ensure greater thermoregulatory

strain for a prolonged period of time. The lower physiological strain in the present study would result in lower metabolic heat production and this may be the reason why a lower level of increase in T_{rec} was observed in the present study during exercise compared to previous research.

During the PT [BLa] appears to show that both trials performed at similar intensities and was not significantly different. However, as noted by Tucker and Noakes (2009), a recalculation of pacing strategy may have occurred towards the end of the PT which would of masked any differences in [BLa] between trials. Contrast to this idea, pacing strategy during the PT did not significantly differ between trials so therefore can be concluded that both trials performed at similar intensities based on [BLa] values.

5.3 Fluid Balance

Body mass losses and subsequent sweat rates were not significantly different between trials on completion of the seated rest or exercise trial. It is generally accepted that exercise performance is not significantly affected by dehydration between 1-2% of body weight in temperate environments (20-21°C) (Cheuvront, Carter, and Sawka 2003). However, exercise has been shown to be adversely affected by dehydration between 1-2% of body weight in hot environments (>30°C) (Coyle 2004, Cheuvront, Carter, and Sawker 2003). Therefore it is possible that performance was not adversely affected by dehydration in the present study as body mass losses did not reach the values aforementioned. However, if the exercise duration was longer or a higher environmental temperature then greater levels of dehydration may occur which may adversely affect performance.

5.4 Skin Blood Flow

The present study shows that SkBF during cooling did not significantly differ between trials at any time point. A possible reason for this may be due to the cooling method or duration applied not being sufficient to elicit any changes. Bogerd et al. (2010) found that after 45 min of ice vest cooling thigh SkBF decreased by approximately 17% from baseline which is similar to the values observed at the end of cooling in the present study (18%). However, Bogerd et al. (2010) observed SkBF values at the chest and back to decrease by ~40 and ~52%, respectively, which was more pronounced than the current study (2 and 30%, respectively). Bogerd et al. (2010) pre-cooled participants for a longer duration compared to the present study which may have provided an accentuated cooling stimulus causing a significant difference between the cooling and control trials. Despite this, Bogerd et al. (2010) showed that T_{ms} was only reduced by $2.7 \pm 0.7^{\circ}\text{C}$ at the end of cooling which was not as substantial as the present study (3.5 ± 0.5) using the same type of ice vest (Arctic Heat). These results show that lower values of T_{ms} do not lead to lower values of SkBF. Indeed, the lower SkBF values observed in the Bogerd et al. (2010) may be due to the longer duration of cooling applied affecting the hypothalamus control of the cutaneous circulation directly. Wilson et al. (2007) observed that whole body cooling (water perfused suit $15\text{-}18^{\circ}\text{C}$) for 20 min significantly reduced hand (dorsal portion) SkBF over time. However, no control trial was included within this study so therefore cannot compare to control SkBF values. Nevertheless, hand SkBF was reduced by ~55% from baseline at the end of cooling which is a more profound decrease than any site at the end of the cooling period in the present study. It is likely the water-perfused suit provided a greater cold stimulus than the present study. However, T_{ms} at the end of cooling in Wilson et al. (2007) was $\sim 28^{\circ}\text{C}$ which is similar to the present study. It has to be taken into consideration that the water-perfused suit may have been successful in clamping skin temperature to similar values across the body (in the four

measured sites; chest, arm, thigh and calf) at $\sim 28^{\circ}\text{C}$, whereas the present study had dramatically lower back skin temperatures compared to the bicep, thigh and calf. It is possible that this uniform skin temperature across the body appears to have lowered SkBF to lower values observed in the present study.

Values for SkBF did not significantly differ between trials at any time point during exercise and the PT which may be due to an absence in a significant reduction in SkBF during the cooling period. There appears to be a large individual variation in the level of SkBF during exercise which may have caused the large standard deviations which could of masked any potential significant observations. Another possible reason for the large standard deviations may be explained by the sensitivity of the laser-Doppler probes. The laser-Doppler probes measure SkBF by recording the rate of movement of blood cells through the skin of the microvasculature. Any disturbance of the probe, such as during exercise, would potentially record values as changes in SkBF when in fact it is external disturbance (Berardesca et al. 2002). This could be another possible reason for the large standard deviations observed in all sites during exercise which may be why no significance was observed.

The present study shows that SkBF values during exercise in both trials increased significantly over time in all sites measured. Gonzalez-Alonso et al. (1999) observed SkBF values for a non-exercising region of the body (forearm) at 45 min of exercise in the heat to be approximately 260% greater than baseline values in both their cooling and control trials. However, these SkBF values are far lower than a non-exercising region of the body(bicep) observed at any site at 45 min of exercise in the present study which was observed to reach 1183% greater than baseline values . In addition, Gonzalez-Alonso, Mara-Rodriguez, and Coyle (2000) observed that a 0.5°C increase in T_{oes} during exercise (cycling at $72\% \dot{V}\text{O}_{2\text{max}}$

for 30 min) in the heat (35°C) resulted in a ~400% increase in SkBF at the forearm measured via a laser-Doppler. However, these values are still lower than those observed during exercise in the present study. A possible reason for the differences in SkBF between previous research and the present study may be related to the area that is being measured. The research discussed measured SkBF from the forearm but does not account for regional differences.

The greatest increase in SkBF in the present study was observed for the thigh. It was observed that in CON thigh SkBF was significantly greater than back SkBF from 45 min of exercise to the end of the PT. The reason for the significantly greater values of SkBF is that the working muscle would produce considerable metabolic heat which would increase surrounding tissue temperature via heat exchange pathways (Cheuvront and Haymes 2001). The subsequent increase in local temperature would influence local and central control of cutaneous circulation which would provide an efferent response to increase SkBF locally (Charkoudian 2003). These results demonstrate significant regional differences in SkBF between exercising areas compared to non-exercising areas.

Values for SkBF between trials did not significantly differ at any point during the seated rest trial. Again, as aforementioned, it may be due to the absence of a significant reduction in SkBF during cooling that no subsequent differences between trials were observed. As shown in the results, large standard deviations are still evident in each trial during seated rest where the participants remained still. The large standard deviations may be as a result of the individual variation in SkBF responses and not disturbance to the laser-Doppler probe due to excessive movement (e.g. exercise).

The present study also shows that there are significant differences in SkBF between seated rest and exercise. Back SkBF in both the seated rest and exercise trials increased significantly over time, however, back SkBF was significantly greater during exercise when compared to the seated rest trial. The increase in SkBF in non-exercising regions during exercise would maximise the area for convective heat loss to dissipate metabolic heat production which has been previously shown (Nadel 1986, Theisen et al. 2001).

5.5 Core Temperature

Ice vest cooling did not significantly lower T_{aur} or T_{rec} compared to CON during the cooling period. Previous research has noted that pre-cooling utilising ice vests did not significantly decrease T_{rec} (Duffield et al. 2003). Castle et al. (2007) observed that pre-cooling participants using ice vests for 20 min did not significantly decrease T_{rec} on completion of cooling. Similarly, Price, Boyd, and Goosey-Tolfrey (2009) pre-cooled participants for 20 min and, again, found no significant difference in T_{rec} on completion of cooling. It is possible that the cooling duration and modality was not sufficient to produce an immediate significant effect on T_{rec} . However, it has been previously noted that ice vest cooling for 45 min also had no significant immediate effect on T_{rec} (Bogerd et al. 2010). Another possible reason is that substantial vasoconstriction may occur during cooling which would direct blood to the core which would inevitably maintain core temperature thus no immediate effect would be noticeable.

During the seated rest trial in the heat both T_{rec} and T_{aur} significantly differed between trials, however the temporal pattern of response differed between the core sites measured. Values for T_{aur} were significantly lower throughout the seated rest trial following cooling but both trials had an increase in T_{aur} throughout. In contrast to this, T_{rec} following the cooling period

decreased progressively and was significantly lower than CON on completion of the seated rest trial. This difference in the temporal pattern of response between T_{rec} and T_{aur} is possibly due to differences in the relative blood flow and mass between sites (Gagnon et al. 2010). Taking this into consideration, the rectum has a large mass and density of tissues compared to the aural canal, therefore a greater amount of heat is required to increase the temperature that is measured with a rectal probe. In addition, blood flow to the region surrounding the rectal probe (i.e. splanchnic and renal) is substantially reduced during exercise and heat stress (Rowell 1974). For example, renal and splanchnic blood flow during passive heat stress is reduced from $\sim 2.8 \text{ L}\cdot\text{min}^{-1}$ at rest in normothermic conditions to $\sim 1 \text{ L}\cdot\text{min}^{-1}$ during hyperthermic conditions (Rowell 1974) and $0.5 \text{ L}\cdot\text{min}^{-1}$ during moderate to heavy exercise in the heat (Sawka and Pandolf 2010). In comparison, the area surrounding the aural canal temperature probe likely receives a relatively large blood flow relative to its mass. Thus, it is possibly due to this lower relative blood flow and greater mass of the rectum which takes considerably longer for significant cooling affects to be observed in comparison to temperature taken at the aural canal. Blood flow to the aural canal is possibly maintained during seated rest in the heat and thus the cooler blood from the cutaneous circulation, as a result of ice vest cooling, is having a quicker affect on T_{aur} compared to T_{rec} . The results of the present study demonstrate that the cooling effects of wearing an ice vest are not lost immediately upon removal of the ice vest as shown through the T_{aur} and T_{rec} values. Therefore, it is possible that an exercising individual may have to complete ice vest cooling 45 min prior to exercise so that there is a significant decrease in T_{rec} and T_{aur} at the start of exercise.

During exercise T_{aur} was significantly lower from 5-25 min following the cooling period compared to CON but no significant differences were observed thereafter. However, T_{rec} did

not significantly differ between trials at any time point during exercise or the PT. As discussed, T_{aur} is more responsive to change in core temperature in comparison to T_{rec} . It appears that these results are similar to the seated rest trial in relation to the quicker response to change in temperature at the aural canal. However, as the blood flow at the rectum is lower during exercise (Sawka and Pandolf 2010) the potential cooling provided by the ice vest does not translate to lower T_{rec} possibly due to the heat sink at the skin being reduced with time during exercise and therefore resulting in no significant difference between trials.

Price, Boyd, and Goosey-Tolfrey (2009) observed a significant difference in T_{rec} between cooling and control trials during exercise from 35 min onwards. Values at 35 min for T_{rec} in the control trial had increased by 1.2°C from baseline which is considerably greater than the 0.5°C rise in T_{rec} at the same time point in the present study. In addition, Duffield et al. (2003) observed an increase in T_{rec} of 1.1°C from baseline in a control after 40 min of intermittent exercise. It is possibly due to the exercise protocol utilised in the present study not providing a substantial thermoregulatory strain on the participants compared to previously used exercise protocols. Despite this, participants were exercising at 73% of their HR max. The physiological responses, such as $SkBF$, to a greater exercise load may have differed from the present study which is a possible direction for future research.

Values for T_{rec} on completion of 45 min of exercise were on average 0.7°C greater than those observed on completion of the seated rest trial. Values for T_{aur} were 1.1°C greater on completion of 45 min of exercise compared to on completion of the seated rest trial. The reason for the greater core temperatures during exercise is due to the greater metabolic heat production compared to seated rest in the heat. It is possibly due to the greater metabolic heat

production during exercise that no significant differences in T_{aur} were noted after 25 min of exercise as the cooling effects of the ice vest appear to have diminished thereafter. This is in comparison to the seated rest trial as T_{aur} remained significantly lower following cooling throughout which again shows the thermoregulatory benefits of wearing an ice vest are not lost immediately.

Despite no significant differences for SkBF between trials, values for T_{aur} were lower following the cooling period during exercise compared to CON. These results further the mechanism proposed by Price, Boyd, and Goosey-Tolfrey (2009). It is possible that the ice vest cooling simply provided a significant heat sink. The greater heat sink at the skin may have provided a greater thermal gradient between the blood and skin which would lead to the blood perfusing the cutaneous circulation being cooled. Thus, the blood returning the core is cooler and thereby abating the rise in core temperature.

5.6 Skin Temperature

Values for T_{ms} on completion of cooling were significantly lower compared to CON.

Previous research has also been successful in lowering T_{ms} using ice vests (Price, Boyd, Goosey-Tolfrey 2009, Bogerd et al. 2010) but others have been unsuccessful (Castle et al. 2007). A major disadvantage with using ice vests is that they are not tailored to an individual which may reduce the heat transfer efficiency (Bogerd et al. 2010). Indeed, Webster et al. (2005) found that ice vests that were designed to be a tighter fit to the skin in comparison to a looser fitting vest resulted in a lower T_{ms} during cooling. The tighter fitting vest also reduced thermal sensation during exercise in the heat (without ice vest) and produced a more rapid decrease in T_{rec} during recovery (with an ice vest). It was clear in the present study that the ice vest did not fit tight to the skin on participants that had an ectomorphic body type in

comparison to participants who had a mesomorphic body type. Needless to say this would have affected the heat transfer efficiency between the vest and skin. Perhaps this is partly the reason why previous research may not have found any significant effect of ice vest cooling on T_{ms} as the ice vest may have not fitted well on participants.

Individual skin temperature sites show that on completion of the cooling period the skin temperature at the back and bicep was on average 9.0 and 2.0°C, respectively, lower compared to CON. The larger heat sink is located in the upper body which is due to the proximity of where cooling was applied. The present study also offers reliability data in relation to the use of ice vest cooling. It was shown that T_{ms} and T_{back} were reduced consistently on both occasions where participants were cooled. It is paramount that the cooling procedure being utilised is replicable to ensure that accurate and reliable data is collected as done so in the present study.

No significant differences in T_{ms} were noted between trials throughout the seated rest trial in the heat with both trials experiencing a continuous rise in T_{ms} throughout. The reason for the increase in skin temperature is due to the thermal gradient between the environment and the skin. As the environment was maintained at 35°C this would result in participants experiencing conductive heat transfer as skin temperature was considerably lower than the environmental temperature in both trials at the beginning of the seated rest trial in the heat. Despite no significant differences between trials for T_{ms} , values for T_{back} were the only skin site to remain significantly lower compared to CON at the end of the seated rest trial. Thus, the maintenance of a significant heat sink in the upper body may have contributed greatly to the reduction in T_{rec} significantly at the end of the seated rest trial compared to CON.

Values for T_{ms} were similar between trials throughout exercise and the PT. Although T_{ms} did not significantly differ between trials, values for T_{back} remained significantly lower for 15 min of exercise following cooling compared to CON. It is possible that this significantly lower heat sink may be the main reason for the significantly lower T_{aur} for 25 min during exercise.

Despite exercise causing an increase in metabolic heat production T_{ms} was only 0.3°C greater on completion of 45 min of exercise compared to completion of the seated rest trial. The blunted rise in T_{ms} during exercise may have been attributable to the substantially greater SkBF values observed in the present study during exercise providing a greater heat exchange between the environment and the skin.

5.7 Readiness to Exercise & Rating of Perceived Exertion

It was shown that RTE was not significantly different between trials which was rated as "partly ready" at all time points which demonstrates that ice vest cooling did not have any significant psychological effect on RTE. This may be possibly due to the lack of significant difference between trials for T_{aur} and T_{rec} . If there was a significant difference between trials in core temperature measurements then this may have affected the participants psychologically and thus may have affected their readiness to exercise. Values for T_{ms} differed between trials during cooling which shows that RTE appears to be unaffected by skin temperature. A decrease in core temperature may have provided the stimulus needed for the participants to be "completely ready" to exercise in the heat.

It was also reported that RPE on completion of 45 min of exercise was rated as "hard" in both trials which was supported by HR values on completion of exercise ($147 \pm 12 \text{ beats} \cdot \text{min}^{-1}$) in

addition to a high correlation between HR and RPE throughout exercise ($r = 0.98$).

Participants in both trials rated their RPE as 18 ± 1 on completion of the PT, which combined with HR values suggests that participants were at or very close to maximal exertion.

5.8 Profile of Mood States

Scores for the POMS did not significantly differ at any time point between trials during exercise and the seated rest trial. However, there was tendency for the "fatigue" subscale to significantly differ between trials ($P=0.08$). These results show that ice vest cooling had a tendency to reduce the psychological feeling of fatigue compared to CON during exercise.

In addition, only the "fatigue" subscale significantly differed over time. Previous research has noted that POMS significantly differ more between pre-to-post exercise where greater dehydration is present (Casa et al. 2010). Therefore, as the present study did not observe any significant differences in body mass losses between trials this may be a possible reason why no significant differences in POMS were observed. The present study had similar results to that observed by Duffield et al. (2003). Duffield et al. (2003) observed that cooling prior to exercise in hyperthermic conditions did not improve participants reported scores on a POMS questionnaire in comparison to a control group. Similarly to the present study, Duffield et al. (2003) did not witness a decrease in rectal temperature after cooling in comparison to the control group. The lack of decrease in rectal temperature with cooling prior to exercise may be the reason why participants did not experience an improvement psychologically within the study.

5.9 Rating of Perceived Thermal Sensation

Not surprisingly, RPTS were significantly lower throughout the cooling period compared to CON. The present study shows that ice vest cooling was again reliable and consistent in reducing RPTS during cooling. No other time point significantly differed between trials in both the exercise and the seated rest trial. These results show that although participants felt cooler while wearing an ice vest the psychological affects did not lead to any significant improvements in exercise performance which is consistent with previous research (Duffield et al. 2003).

In both trials RPTS throughout the exercise and the seated rest trials had a greater positive correlation with T_{ms} ($r = 0.98$) compared to T_{aur} ($r = 0.92$) and T_{rec} ($r = 0.80$). Therefore, the feeling of how warm an individual feels appears to be related more to T_{ms} and less to T_{aur} or T_{rec} . Previous research has also shown thermal comfort to improve with significantly lower T_{ms} following ice vest cooling where there were no reductions in T_{rec} (Hornery et al. 2005, Duffield et al. 2003). However, this could be potentially unsafe as an exercising individual may perceive themselves as feeling more “comfortable” during exercise preceded with cooling and therefore may exceed their critical core temperature (Tucker and Noakes 2009).

5.10 Performance Trial

No significant difference for performance was observed between trials. However, six out of eight participants improved their performance time following ice vest cooling. Those who improved their completion times following the cooling period had an average improvement of 23 seconds ($n = 6$). However, those who did not improve their performance time were on average 43 seconds slower following the cooling period ($n = 2$). Upon looking at the data between the two groups (participants that did improve and participants that did not), those who did not complete the PT faster following the cooling period did not experience any

significant decrease in T_{ms} during cooling. It is possible that the participants who did experience a significant effect of ice vest cooling on T_{ms} may have been psychologically more prepared for the PT. Values for RTE were greater prior to the PT for those who improved in the PT compared to those who did not (4.5 ± 0.8 and 3.8 ± 0.4 , respectively). In addition, those who did not improve in the PT perceived themselves as more fatigued at the end of the 45 min of exercise as shown in the "fatigue" subscale in the POMS compared to those who did improve in the PT (2.5 ± 1.0 and 1.7 ± 0.7 , respectively). Therefore, it is possible that the significantly lower T_{ms} observed during cooling had a positive psychological affect on the participants which resulted in faster completion times.

Goosey-Tolfrey et al. (2008) observed that completion times of a 3-km time-trial cycle preceded by 60 min of exercise in the heat (30.8°C) was significantly improved by 14 seconds in a hand cooling trial (10 min at 10°C applied immediately prior time-trial) compared to a control. Completion times in Goosey-Tolfrey et al. study were 267 and 281 seconds for the cooling and control trials, respectively, which were similar to the completion times in the present study. Despite Goosey-Tolfrey (2008) and the present study recruiting a participant population not representing highly trained individuals (49.3 and $43.4 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$, respectively), it appears that well trained cyclists ($\text{VO}_{2\text{peak}} = 60.1 \text{ ml}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$) completed a 3-km time-trial in 266 seconds (Hajoglou et al. 2005) which is similar to both Goosey-Tolfrey et al. (2008) and the present study. The pacing strategy, as shown through the change in cadence, did not significantly differ between trials in the present study, however there was a significant increase across time in cadence from 2.5-km to 3.0-km which could represent a recalculation in pacing strategy towards the end of the PT (Tucker and Noakes 2009).

6.0 Conclusion

In conclusion, this study intended to investigate the effects of pre-cooling on skin blood flow responses during exercise and seated rest in the heat. From the results observed the present study furthers the mechanism proposed by Price, Boyd, and Goosey-Tolfrey (2009). It was hypothesised that SkBF would be significantly reduced during the cooling period compared to a control of no cooling which would lead to significant differences between trials during subsequent exercise and seated rest. Values for SkBF were not significantly reduced during cooling nor were attenuated compared to no pre-cooling during subsequent exercise or seated rest in the heat. Therefore the hypothesis that a cooling period would significantly affect SkBF was not met. However, the mechanism in which pre-cooling effects core temperature may relate to the significant heat sink created in T_{ms} compared to CON. As SkBF was maintained following the cooling period and while significantly reducing T_{ms} the blood perfusing the cutaneous circulation during subsequent exercise or seated rest in the heat would be cooled more so compared to a no cooling trial. The cooler blood would then attenuate a rise in core temperature on return as shown in T_{aur} during exercise the present study. The seated rest trials show that SkBF increased over time but was significantly lower than that observed during exercise in a non-exercising region (back).

It was also hypothesised that cooling would not have an immediate significant effect on core temperature but would significantly differ during exercise and seated rest. The results show that both T_{aur} and T_{rec} were significantly lower during the seated rest trial following a cooling period compared to CON, although it did take 45 min of seated rest in the heat for T_{rec} to

significantly differ. Only T_{aur} was significantly different during exercise following a cooling period compared to CON which is possibly due to the differing temporal pattern of response at different core sites measured (Gagnon et al. 2010). Therefore this hypothesis was fully supported for the seated rest trial, although only partially supported for the exercise trial as T_{rec} did not differ during exercise following cooling. Ice vest cooling in the present was reliable in reducing T_{ms} , T_{back} and RPTS. It is important that the cooling protocol being utilised is replicable to ensure valid results. Results for the POMS questionnaire and RTE showed no significant differences between group.

Performance was not significantly improved compared to CON. No physiological or psychological measure demonstrated improvements with ice vest cooling. Future research should assess what the optimum time to perform exercise after ice vest cooling as it was shown in the present study T_{rec} only significantly differed after 45 min of seated rest in the heat following cooling.

7.0 Limitations and Future Research

The sample size of the current study ($n=8$) was also a limitation as it was only a small representation of group sample. If the sample size was larger in the present study then a statistical difference may have been obtained in those parameters that were approaching significance. A statistical power analysis test was performed post-hoc which revealed that at least 71 participants would be needed across two groups to appropriately power the present study to obtain statistical significance. In addition, the sample of the present study is not representative of the participants that would potentially undertaking prolonged exercise during hyperthermic conditions. Future research should recruit highly fit individuals ($>65 \text{ ml.kg.min}^{-1}$) as, although, participants appeared to give full effort, a highly fit group would be more appropriately motivated to exercise in addition to being enabled to reach greater core temperatures (Nielsen et al. 1990). Another limitation of the study is that participants were either completing anaerobic or aerobic training. Which meant that those participants who were completing anaerobic training could of potentially experienced greater core temperature during the 45 min of exercise but would of performed well during the PT. In contrast to this, those participants who were completing aerobic training potentially could of had lower core temperatures during the 45 min of exercise but may have performed poorly during the PT.

The method of cooling utilised in the present study, although considered applicable to field settings, did not produce a reduction in SkBF during cooling and attenuation during exercise although T_{aur} was reduced during exercise. In contrast to this, Bogerd et al. (2010) showed that 45 min of ice vest cooling significantly reduced SkBF at various sites measured. In

addition, Wilson et al. (2007) observed a larger decline in forearm SkBF after 20 min of cooling (water perfused suit) than in any site measured in the present study. It is possible that the present study did apply cooling for a long enough duration to elicit any significant differences in SkBF. In addition, a more uniform skin temperature found in cooling methods such as water perfused suits may be required to reduce SkBF during cooling.

The values for the increase in T_{rec} and [BLa] during exercise were lower than that previously found during intermittent exercise (Price, Moss, and Rance 2003, Price, Boyd, and Goosey-Tolfrey 2009). This shows that the present study did not provide substantial thermoregulatory strain compared to previous research. Future research should try and provide the same levels of thermoregulatory strain as previous research in an attempt to produce any different responses to the present study. A protocol should be developed that is longer in duration so participants would experience greater thermoregulatory strain. In addition, the exercise intensity could also be manipulated. Pilot work should be included prior to testing to obtain the optimum data from an appropriate protocol.

In addition, no fan was utilised in the present study which meant that wind speeds were not replicated to what participants would be exposed to during actual field based exercise. Indoor experiments do not adequately provide the same air movement around a cyclist which is found during field conditions due to the high velocity of cycling (Gagge, Herrington, and Winslow 1937 cited in Nybo 2010: 72). In flat terrains average cycling speed is usually well above 10 m/s which would elevate the heat loss capacity to greater levels than that found in indoor stationary cycling. Lab experiments that attempt to replicate outdoor cycling by utilising fans will typically generate wind speeds that are much lower than actual field conditions (Nybo 2010). This would result in a difference in environmental conditions

experienced at the same ambient temperature. Thermal balance may be established at the same relative intensity and ambient temperature during outdoor cycling compared to laboratory conditions due to the high air velocity which is able to facilitate evaporation which may result in a heat stress becoming compensable.

Based on the seated rest results of the present study, future research should intend to find the optimum time in which exercise should be performed after completion of ice vest cooling. The reason for this is that T_{rec} was significantly lower only after 45 min of seated rest in the heat preceded with cooling. Therefore, no immediate affect was found in T_{rec} and thus individuals who are immediately exercising post-cooling may not be performing at an optimum time. Future research could include a protocol that initiates exercise at different time points after cooling with one time point 45 min after cooling. The rationale for this is that the present study observed T_{rec} to be significantly lower only after 45 min of seated rest in the heat preceded with cooling.

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Appendix 1

Ethical Approval

REGISTRY RESEARCH UNIT ETHICS REVIEW FEEDBACK FORM

(Review feedback should be completed within 10 working days)

Name of applicant: Matt Maley **Faculty/School/Department:**
HLS/BSS.....

Research project title: The effects of pre-cooling on skin blood flow during exercise in the heat and subsequent performance

Comments by the reviewer

1. Evaluation of the ethics of the proposal:

The ethics proposal is sound and well written. However, i am not convinced that the responses in section 8 are appropriate. To be on the safe side i would think it better to state that there is potential that the protocol described 'could' result in significant physical harm and may result in the applicant placing participants in situations where harm could be done. I acknowledge that this is unlikely but by virtue of the fact that the protocol involves the researcher asking the participant to perform maximal exercise and then to exercise in a thermal environment, it is probably better to clarify that harm could be done and then explain that this is likely due to unforeseen injury during the experimental protocols. They should then state what safeguards are in place to manage this risk (e.g., use of trained researchers, participants familiar with maximal/high-intensity exercise, use of pre screening questionnaire and physiological monitoring during the test to monitor the participant whilst performing).

A references list should also be provided for those papers/sources cited in the text

2. Evaluation of the participant information sheet and consent form:

The participant information sheet is well written and appropriate. However, it may be useful to include a couple of lines regarding independent information sources that participants might want to consult if they want further information. E.G. The British Association of Sport and Exercise Sciences website or similar.

Consent form is wholly appropriate.

3. Recommendation:

(Please indicate as appropriate and advise on any conditions. If there any conditions, the applicant will be required to resubmit his/her application and this will be sent to the same reviewer).

☐

Approved - no conditions attached

☒

Approved with minor conditions (no need to resubmit)

☐

Conditional upon the following – please use additional sheets if necessary (please re-submit application)

☐

Rejected for the following reason(s) – please use other side if necessary

☐

Further advice/notes - please use other side if necessary

Name of reviewer: Dr Mike Duncan

Signature: M. Duncan

Date: 13/1/11

Appendix 2

Participant Information Sheet

Title: The effects of pre-cooling on skin blood flow during exercise in the heat and subsequent performance

Introduction: You are being asked to take part in a research study conducted at Coventry University. It is paramount that you, the participant, understand why the research is being conducted and what it will involve. Please take time to read the following information carefully. Please do not hesitate to ask the researcher, Matthew Maley, if there is anything unclear to you.

What is the purpose of this study?: The data gained from this study will be used to assess what effect commercially available ice vests have on physiological responses to exercise and performance.

Why have I been chosen?: You have been asked to take part in the present study as you are physically active and a healthy individual.

Do I have to take part?: It is up to you if you decide whether you wish to take part. If you do decide to take part you will be given this information sheet to keep and will be asked to sign a consent form. If you wish to take part in the study you are free to withdraw at any time and without giving a reason. A decision to withdraw at any time, or a decision not to take part, will not affect your marks on your course or the way in which staff treat you.

What will happen to me if I take part?: Your participation in this study will involve six visits to the laboratory. All sessions will last approximately two to two and a half hours including rest, warming up and cooling down.

The first trial will be a test for maximal oxygen uptake ($\text{VO}_{2\text{max}}$) on a cycle ergometer in an air conditioned lab with temperature regulated between 19-20°C. This test will include cycling on the ergometer with an initial power output of 70 W at 70 rpm. Increases of 0.5 Kg will be added every 3 minutes until you are unable to continue exercise at the given cadence. Expired gas will be continually sampled via a breath by breath system (Metamax) in order to determine ventilation rate (VE), oxygen consumption (VO_2), carbon dioxide production (VCO_2) and respiratory exchange ratio (RER). Heart rate (HR) will be continually monitored via a polar heart rate monitor placed around your chest. Rating of Perceived Exertion (RPE) will be recorded during each stage from a scale reading values of six (no exertion) to 20 (maximal exertion). This relates to your personal perceptions of fatigue in your body overall. Fingertip blood samples for analysis of blood lactate (BLa) will be measured before and after exercise.

After the preliminary trial there will be **one familiarisation trial** and **four experimental trials** consisting of two exercise trials and two seated rest trials. One exercise trial will require you to wear an ice vest for 20 minutes prior to exercise. The second exercise trial will be the control as you will not wear an ice vest prior to exercise. The seated rest trials require you to remain seated for the same length of time as the exercise trials (45 minutes). One seated rest trial will require you to wear an ice vest for 20 minutes prior to seated rest

without the ice vest. The other seated rest trial will again be a control where you do not wear an ice vest and rest for the remainder of the trial. The reason for conducting seated rest trials is to provide data on the effects of the ice vest without an exercise stimulus.

Trials will be no less than a week apart to ensure there is adequate rest between trials and to prevent heat acclimation. At the start of each experimental trial you will be required to have ten minutes of seated rest in an air conditioned laboratory with the temperature regulated between 19-20°C. During this time baseline measurements will be taken which will include VO_2 which will be collected via expired gas using a face mask. Skin temperature will be measured using skin thermistors taped to five different sites which include; chest, upper back, upper arm, thigh and calf. Core temperature will be measured from rectal and aural sites. HR will also be continually measured along with skin blood flow which will be measured using a Laser-Doppler which will be attached to four skin sites which include; chest, upper back, upper arm, and thigh. Thermal perceptions, mood states, and readiness to exercise will be collected from a short questionnaire.

You will then receive an ice vest to wear, dependent upon trial, in which you will have seated rest for 20 minutes while baseline data is again collected. After this initial 20 minutes the ice vest will be removed, if you are wearing one, and will then enter the environment chamber where temperature will be 35°C with a relative humidity of 40-60%. You will then either continue seated rest or proceed to exercise on a cycle ergometer. During the exercise trial you will be required to complete an intermittent exercise protocol at a range of intensities between 40-100% $\text{VO}_{2\text{max}}$ obtained in the preliminary trial which will last 45 minutes. This will include 2 min 30 seconds at 40% $\text{VO}_{2\text{max}}$, 1 min 30 seconds at 60% $\text{VO}_{2\text{max}}$, 30 seconds at 100% $\text{VO}_{2\text{max}}$, and 30 seconds unloaded. This will be repeated nine times until 45 minutes of exercise is reached. During the exercise and seated rest trial measurements such as VO_2 , skin and core temperature, HR, skin blood flow, BLa, sweat rate, RPE, thermal perceptions, mood state, and readiness to exercise will be collected.

Following this 45 minute bout of exercise there will be a time trial. This will require you to complete a 3-km distance on a cycle ergometer at 60% $\text{VO}_{2\text{max}}$ as quick as you can. The same measurements will be taken during this time as in the intermittent workload. If you are completing the seated rest trials then you will not have to complete the time trial.

Possible risks involved in taking part: As you will be exercising to $\text{VO}_{2\text{max}}$ on the first trial you are likely to be exercising hard for 2-4 minutes and maximally for 2-3 minutes. An appropriate warm up and cool down will be completed to mediate risk of injury / muscle soreness. Two other trials will require you to exercise in the heat, even though the intensity is not continuously high, there is still the possible risk of heat exhaustion during exercise, however you will be continuously supervised throughout all trials. All subjects will be required to wear appropriate clothing for testing trials (T-shirt, shorts, and trainers) and will be required to complete a standard laboratory risk assessment form and health screen form prior to each trial.

Possible benefits involved in taking part: The benefit to you is that you will have an assessment of your aerobic fitness / performance. You will also gain an insight on how a commercially available ice vest works and how this may affect your performance in the heat. The information obtained from this study will help improve our understanding of how pre-cooling effects skin blood flow during exercise in the heat and subsequent performance.

Confidentiality: Any data from the research will be kept in a secure location with information only available to the Director of Studies; Mike Price, the experimenter; Matthew Maley and the participant themselves. Data will not be shared amongst the participants. Any data stored on computer will be protected by a password and will use participant codes with only Mike Price and Matthew Maley able to access the information. If you have any queries about this research please ask;

Matthew Maley (maleym@uni.coventry.ac.uk; Personal
telephone
number
removed) or

Dr Mike Price (mike.price@coventry.ac.uk; 02476 888163, Room 351 James Starley Building, Coventry University)

Appendix 3

Health Screen Questionnaire

SPORT SCIENCE AND PHYSIOLOGY HEALTH SCREEN QUESTIONNAIRE

This form **MUST** be completed **EACH TIME** that a subject takes part in any tests. The form must be kept on file for 12 months after the date of testing (Files kept in Sport Science Prep Room).

THE PURPOSE OF THIS FORM IS TO CHECK THAT IT IS SAFE FOR YOU TO TAKE PART IN TESTS TODAY. INFORMATION WILL BE TREATED IN CONFIDENCE. IF THERE IS INFORMATION THAT YOU DO NOT WISH TO WRITE DOWN YOU CAN DISCUSS IT WITH THE LAB SUPERVISOR IN CONFIDENCE.

REMEMBER YOU ARE FREE TO WITHDRAW FROM TESTING AT ANY TIME

NAME _____

DATE OF BIRTH _____

AGE _____

TESTS PLANNED FOR TODAY (TO BE COMPLETED BY EXPERIMENTER)

GENERAL PHYSICAL FITNESS

How often do you take regular physical exercise ?

- Less than once a week
- Once a week
- Two to three times a week
- More than three times a week

Is your current body weight

- Normal range
- Overweight
- Underweight ?

How long have you been exercising at this frequency ?

- Less than 1 month
- 1-6 months
- more than 6 months

Smoking habits (tick ALL that apply)

- Never smoked
- Gave up more than 1 month ago
- Total years smoked for
- Smoke/ used to smoke less than 20 cigarettes per day
- Smoke/ used to smoke more than 20 cigarettes per day

GENERAL HEALTH

Do you suffer or have you ever suffered from the conditions below ? (give details if yes)

- | | |
|--|--|
| • Heart disease and/ or circulatory problems | • Kidney disease |
| • Diabetes | • Clotting disorders |
| • High blood pressure | • Anaemia or other blood disorders |
| • High cholesterol | • Any other long term medical disorder |
| • Asthma or any other type of lung disease | |

Details _____

Do you regularly take

- Any prescribed medicines
- Any over the counter medicines
- Any other drugs
- Any supplements

Details _____

please turn over and complete reverse of form

Have you ever had past injuries that might be affected by the tests planned for today ?

Is there any other information that might affect your safety/ health in carrying out these tests ?

YOUR HEALTH TODAY

Have you had any of the following health problems in the last few days

- Coughs / colds
- Headaches
- Shortness of breath
- Muscle/ joint pains
- Any other health problems

Do you currently have any of the following symptoms

- Sore throat or blocked nose
- Shortness of breath
- Headache and or dizziness
- Nausea
- Pain in muscles/ tendons/ bones
- Any other feelings/ pains that you do not normally have

Are you pregnant ?

Is there any other information that might affect your safety/ health in carrying out the tests today ?

I confirm that I have given details of any information that may affect my suitability to participate as a subject today. I have also

EITHER read the appropriate lab class schedule and am aware of the tests that will be carried out and any possible adverse effects
OR completed a specific consent form for this experiment which stated the tests to be carried out and any adverse effects.

TICK ONE

<input type="checkbox"/>
<input type="checkbox"/>

SIGNATURE OF SUBJECT _____

AUTHORISED BY (PRINT NAME) _____
(SIGNATURE) _____

DATE _____

Appendix 4

Borg Scale: Rating of Perceived Exertion

6	NO EXERTION AT ALL
7	EXTREMELY LIGHT
8	
9	VERY LIGHT
10	
11	LIGHT
12	
13	SOMEWHAT HARD
14	
15	HARD (HEAVY)
16	
17	VERY HARD
18	
19	EXTREMELY HARD
20	MAXIMAL EXERTION

Appendix 5

Modified POMS

Please rank your feeling giving the following numerical mark.

	Not at all	A little	Moderately	Quite a bit	Extremely
Anger	0	1	2	3	4
Fatigue	0	1	2	3	4
Vigour	0	1	2	3	4
Depression	0	1	2	3	4
Tension	0	1	2	3	4
Confusion	0	1	2	3	4

Anger: Peeved, Grouchy, Annoyed, Furious

Fatigue: Worn out, Exhausted, Fatigued

Vigour: Lively, Active, Energetic, Cheerful

Depression: Unhappy, Sad, Discouraged, Miserable

Tension: Tense, Shaky, On Edge, Anxious

Confusion: Confused, Unable to concentrate, Uncertain about things, Forgetful

Appendix 6

Original POMS

This item has been removed due to third party copyright. The unabridged version of the thesis can be viewed at the Lanchester Library, Coventry University.

Appendix 7

Validity between original and modified POMS

Pearson's rank correlation was carried out on mean scores between the original and modified POMS. The individual scores for the original and modified POMS are shown below in addition to the results of the correlation and individual t-test results.

MODIFIED																
Participant no.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	AVERAGES
ANGER	0	0	1	1	0	1	0	2	1	0	0	0	0	0	0	0
FATIGUE	2	1	3	0	0	2	2	1	1	2	1	1	0	1	1	1
VIGOUR	2	1	2	2	3	1	2	1	1	1	2	2	2	1	2	2
DEPRESSION	0	0	1	0	0	1	0	1	0	0	0	0	1	1	0	0
TENSION	1	1	2	0	1	1	0	1	1	1	1	0	1	1	0	1
CONFUSION	0	0	0	1	1	1	0	2	1	1	1	1	1	1	1	1

ORIGINAL																
Participant no.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	AVERAGES
ANGER	0	0	2	1	0	1	0	2	1	0	0	0	0	1	0	1
FATIGUE	2	1	3	0	0	2	2	1	1	1	2	1	1	1	1	1
VIGOUR	2	1	2	2	3	1	2	1	1	2	2	2	2	1	2	2
DEPRESSION	0	0	1	1	0	1	0	1	0	0	0	0	1	1	0	0
TENSION	1	1	1	0	1	1	0	1	1	1	1	0	1	1	0	1
CONFUSION	0	0	1	1	1	1	0	2	1	1	1	1	1	1	1	1

	<i>r</i> value
Anger	0.90
Fatigue	0.83
Vigour	0.85
Depression	0.90
Tension	0.86
Confusion	0.74

	<i>P</i> value
Anger	0.94
Fatigue	0.79
Vigour	0.36
Depression	0.72
Tension	0.24
Confusion	0.27

Appendix 8

Perceived Readiness to Exercise

1.0	NOT AT ALL READY
1.5	
2.0	NOT YET READY
2.5	
3.0	NEED MORE TIME
3.5	
4.0	PARTLY READY
4.5	
5.0	COMPLETELY READY

Appendix 9

Rating of Perceived Thermal Sensation

0.0	UNBEARABLY COLD
0.5	
1.0	VERY COLD
1.5	
2.0	COLD
2.5	
3.0	COOL
3.5	
4.0	COMFORTABLE
4.5	
5.0	WARM
5.5	
6.0	HOT
6.5	
7.0	VERY HOT
7.5	
8.0	UNBEARABLY HOT